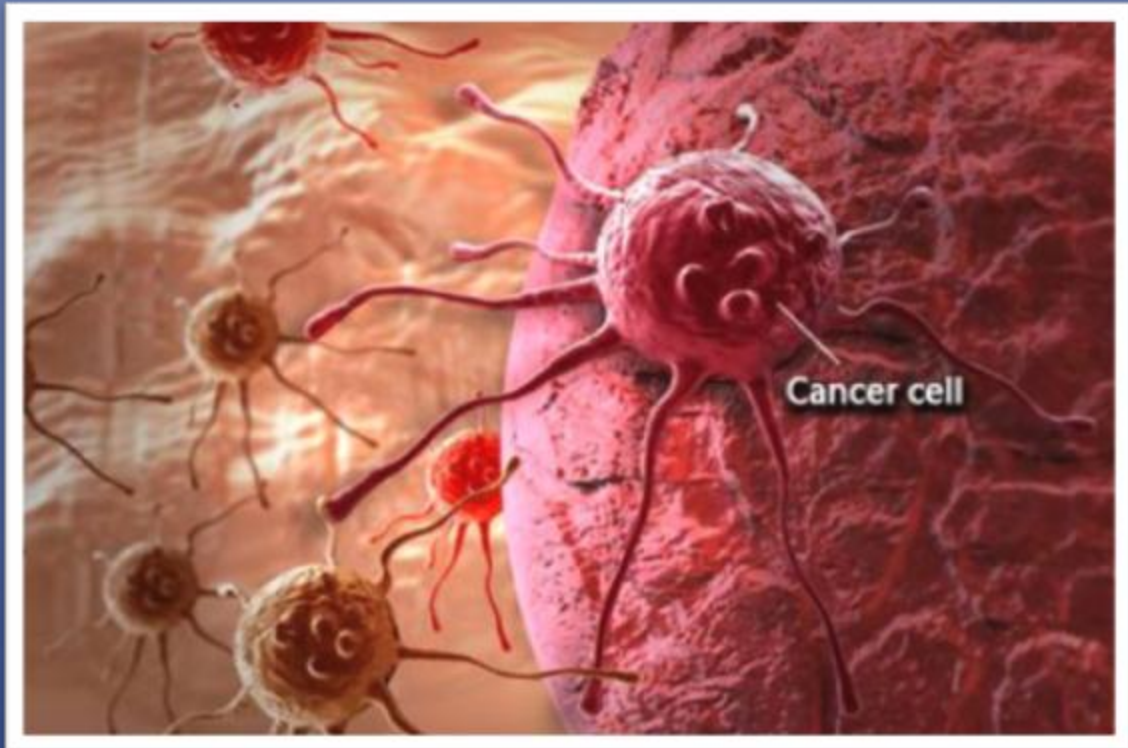


COLOR ATLAS OF GENERAL PATHOLOGY



EDITED BY:

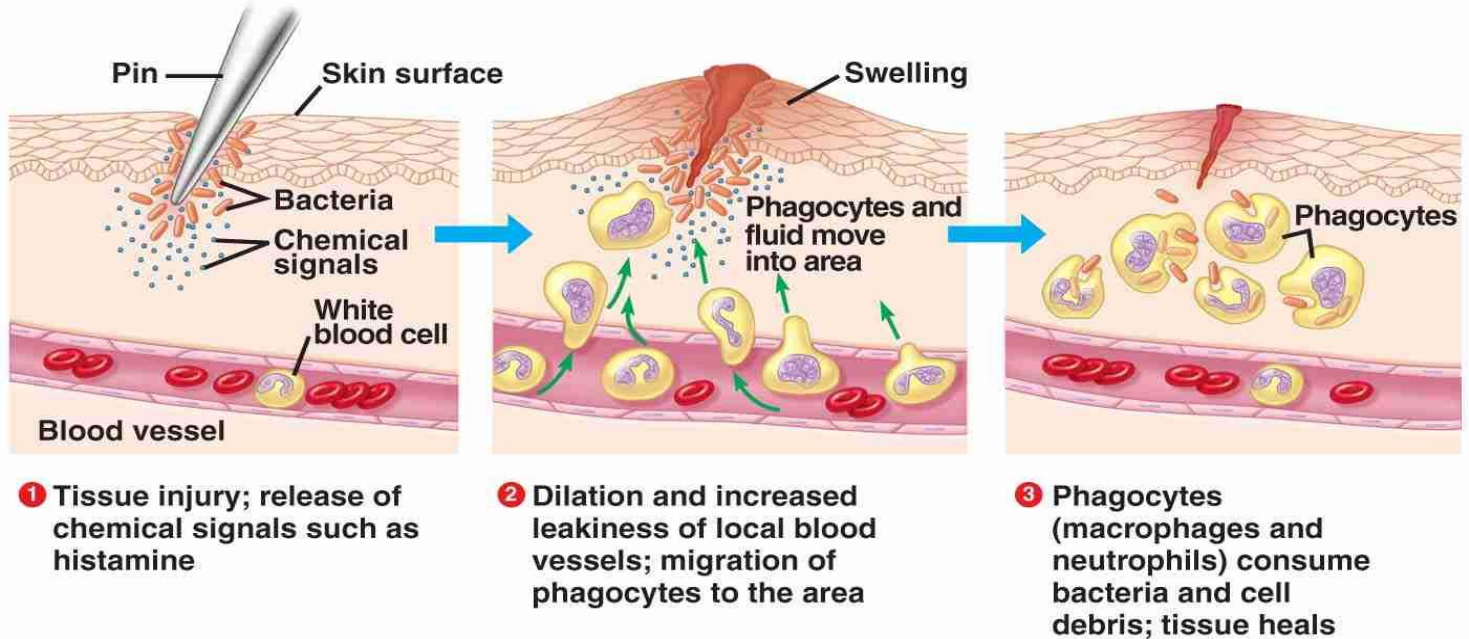
Sherif M. Aboalsaod

FACULTY OF MEDICINE ALAZHAR UNIVERSITY

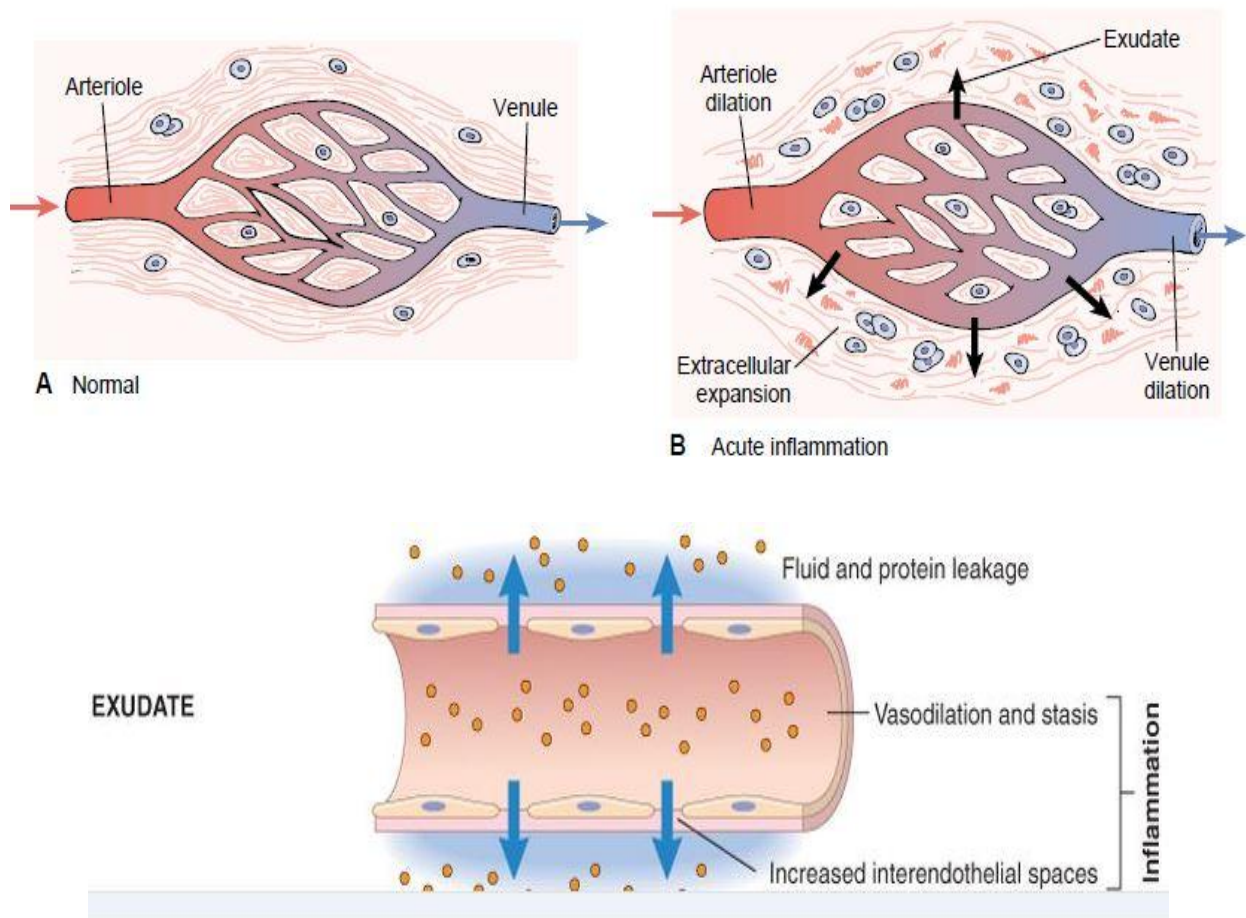
<u>Chapter:</u>	<u>Page</u>
<u>Chapter 1: inflammation</u>	<u>2</u>
<u>Chapter 2: repair</u>	<u>10</u>
<u>Chapter 3: cell injury</u>	<u>17</u>
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Inflammation

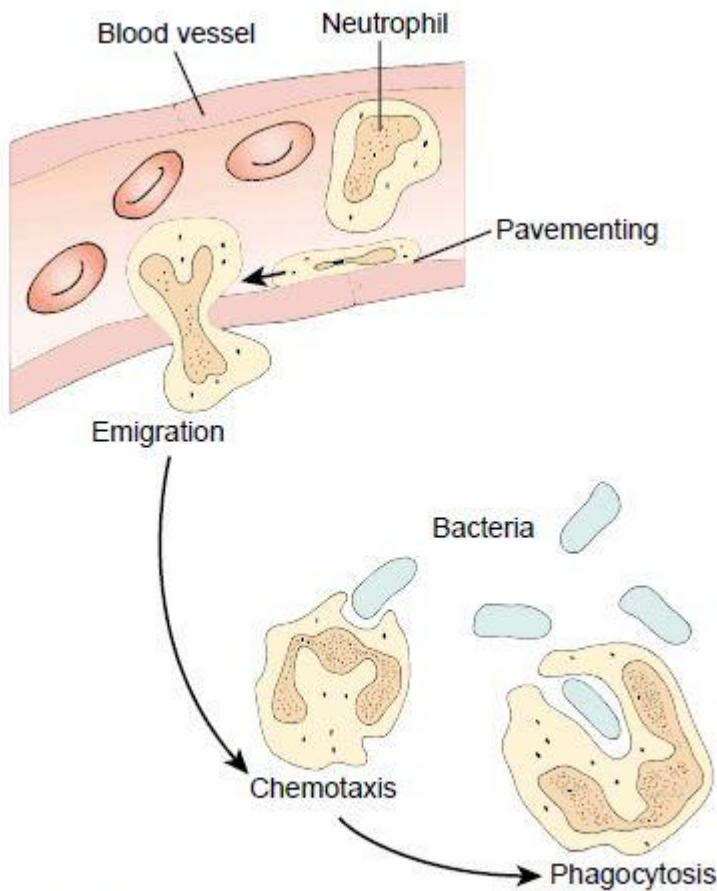
Inflammatory pathway:



Vascular changes and formation of exudate:



Cellular phase of acute inflammation:

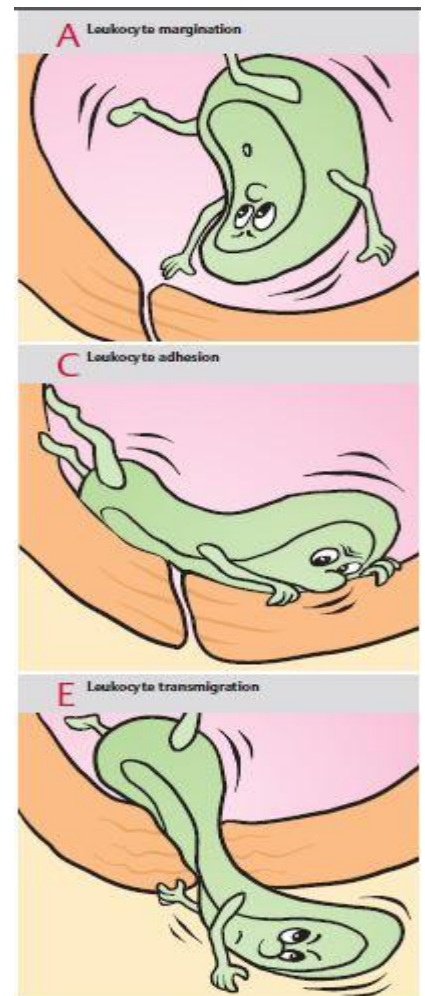


■ **FIGURE 9-3** ■ Cellular phase of acute inflammation. Neutrophil margination, pavementing, chemotaxis, and phagocytosis.

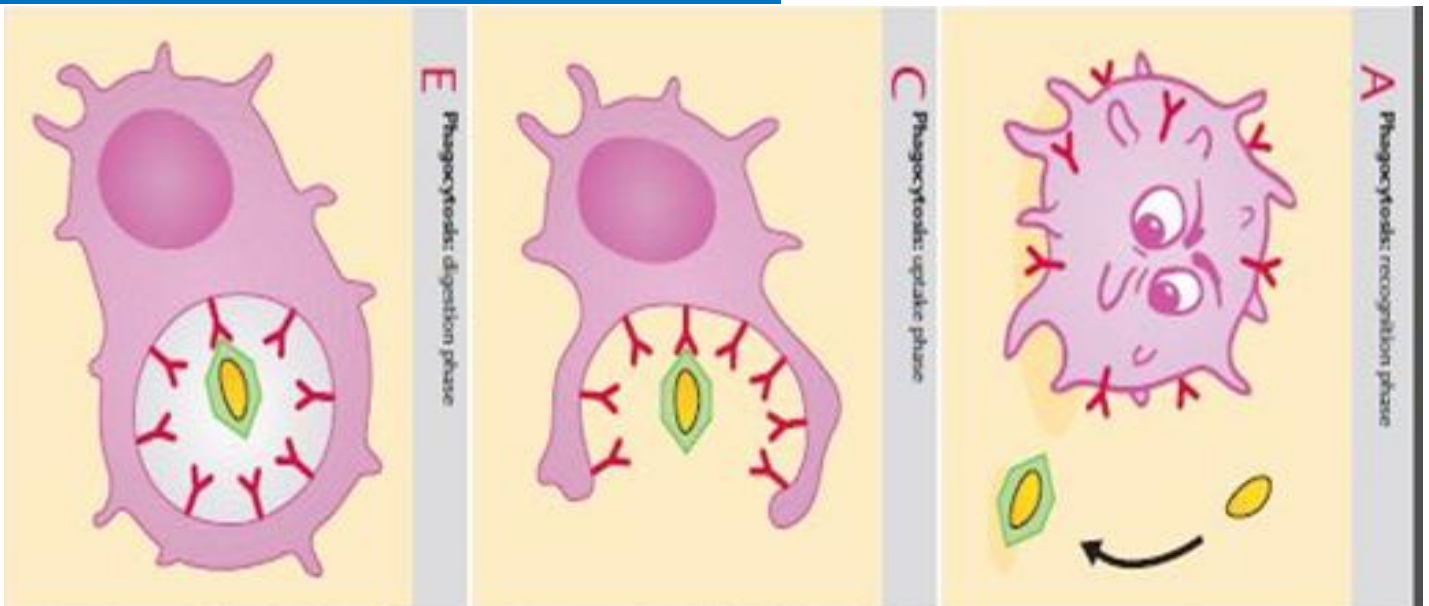
a.margination

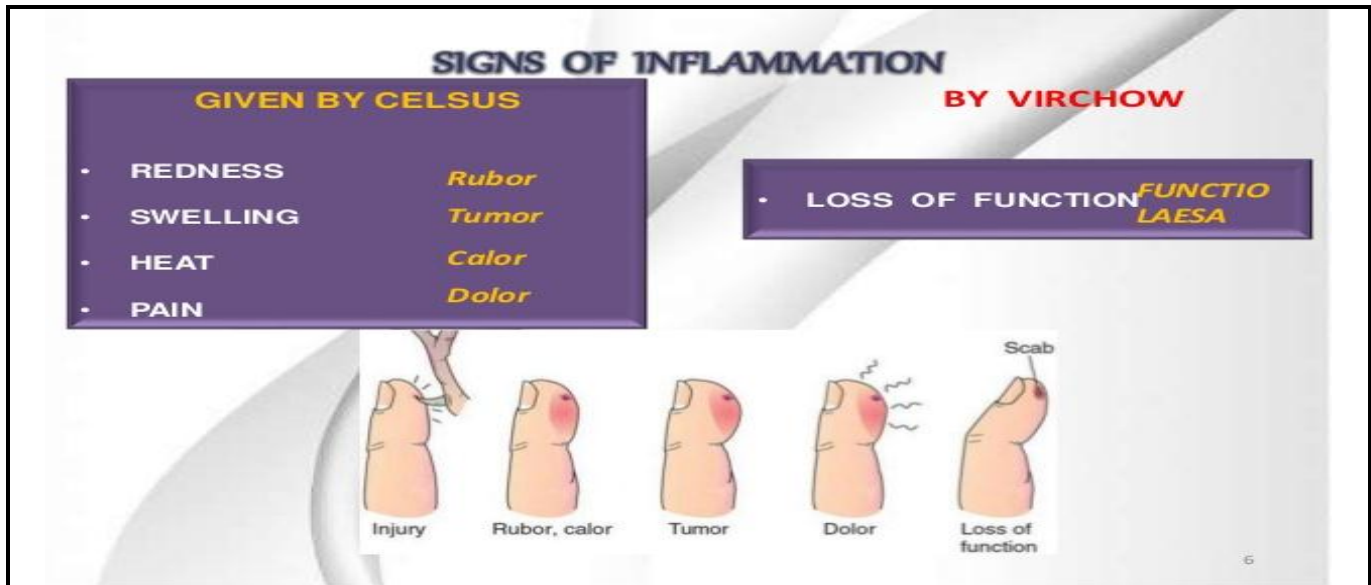
b.pavementation

c.emigration

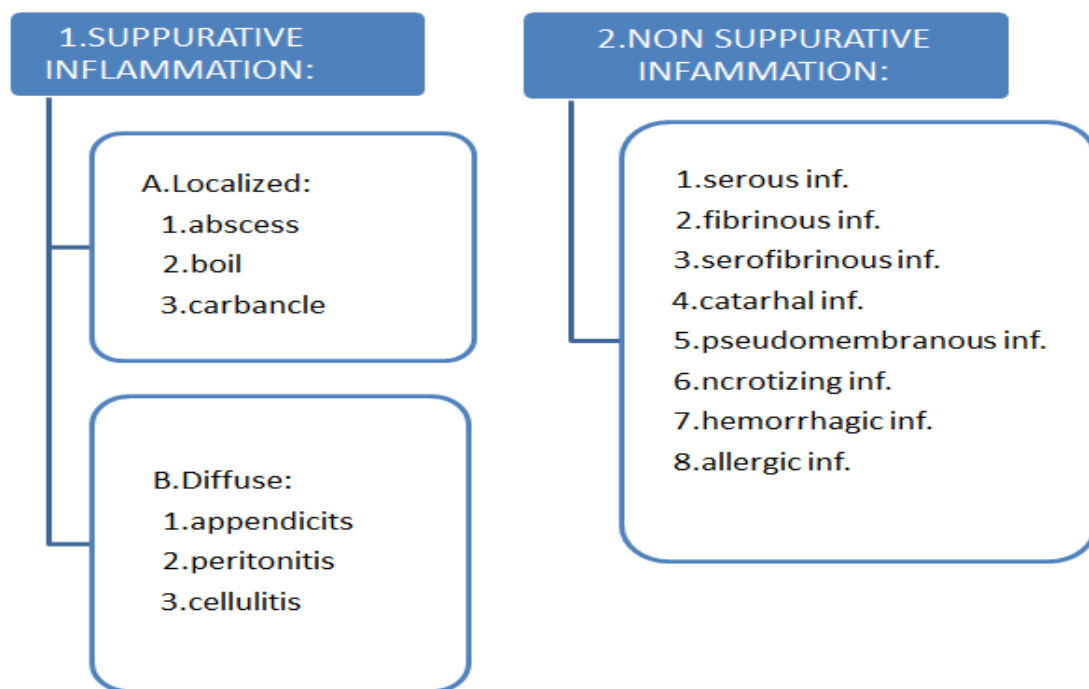


Phagocytosis (recognition >>engulfment>>digestion)

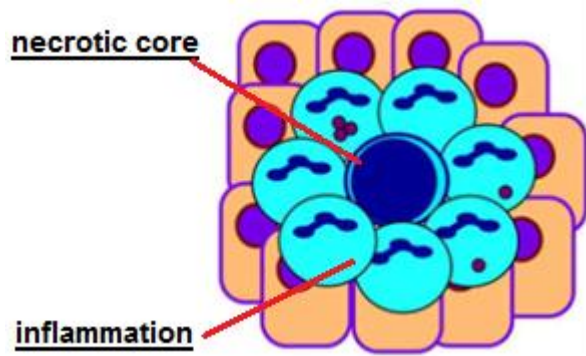




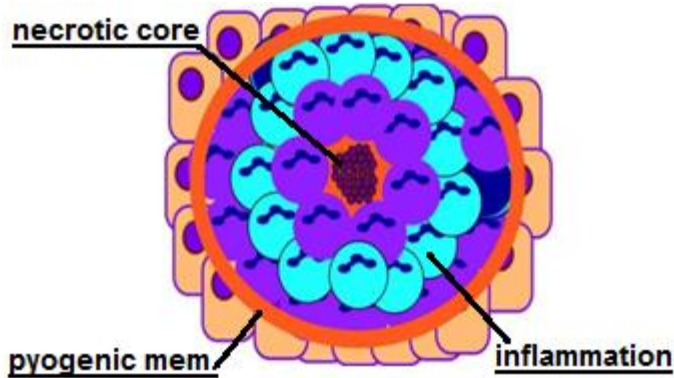
Types of acute inflammation



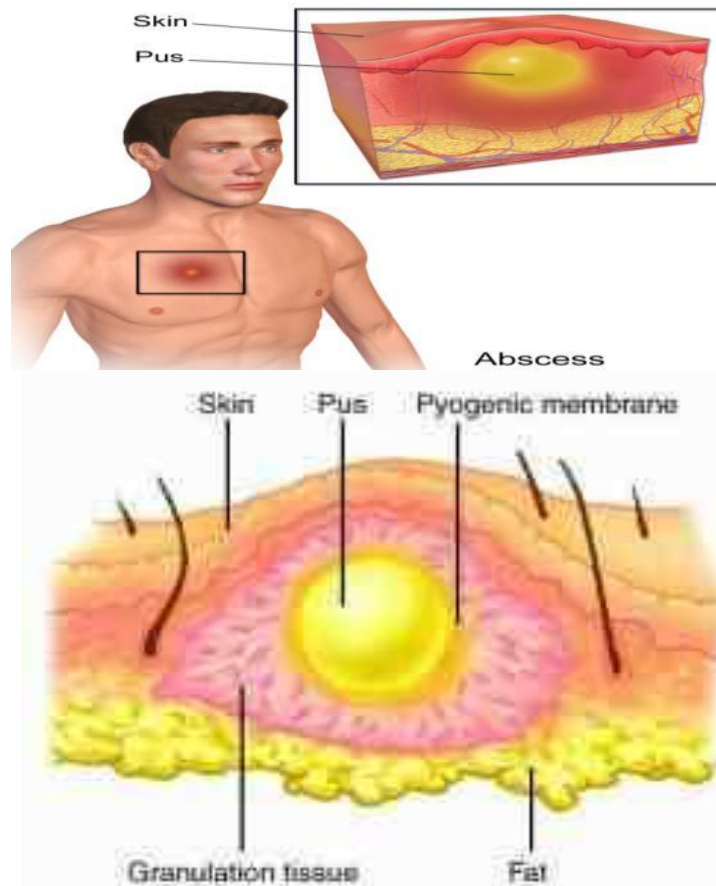
Early 2 zones of abscess:



Late 3 zones of abscess:

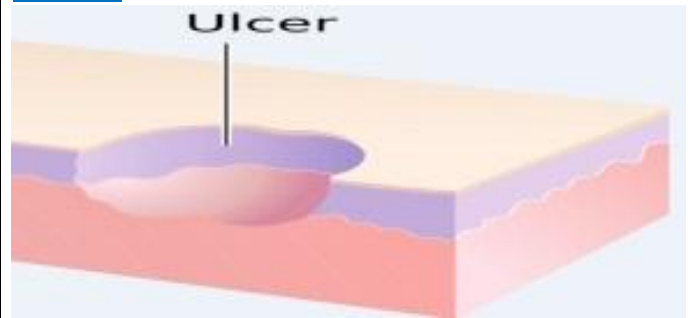


Shape of abscess:



Complication of abscess:

*ulcer:



skin ulcer over patella



*sinus (a) and fistula (b)

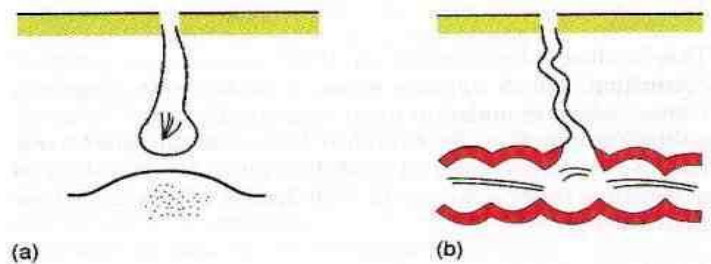
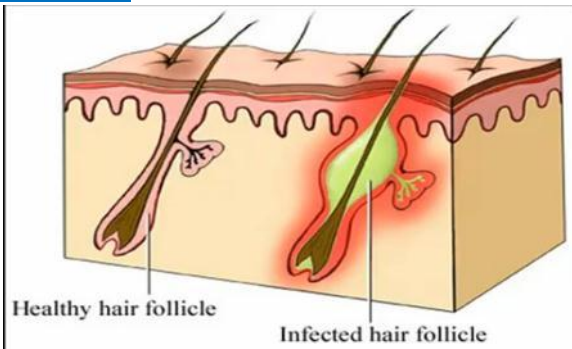


Fig. 12.18 (a) A sinus, and (b) a fistula. Both usually arise from a preceding abscess. (a) This shows that a sinus is a blind track, in this case a pilonidal sinus with its hairs; (b) this shows that a fistula is a track connecting two (epithelial) lined surfaces, in this case a colocutaneous fistula.

keloid

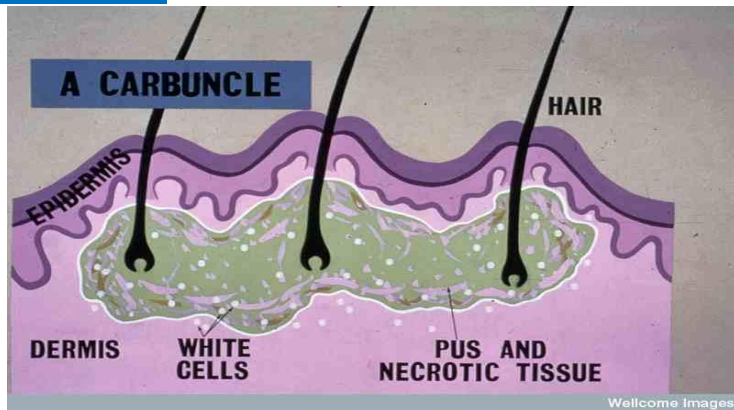


Boil (fruncle):



Boils (Skin Abscess)

Carbuncle:



Carbuncle



1.Serous inflammation:



2.fibrinous inflammation (lobar pneumonia):

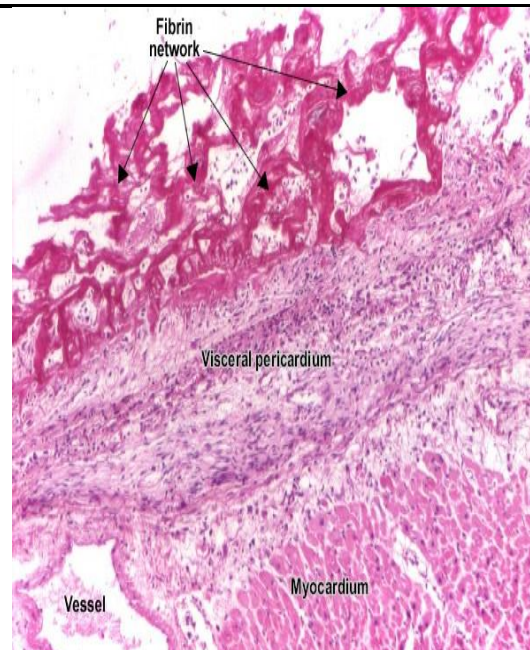


3.serofibrinous inflammation (pericarditis):

Gross picture:



Microscopic picture:

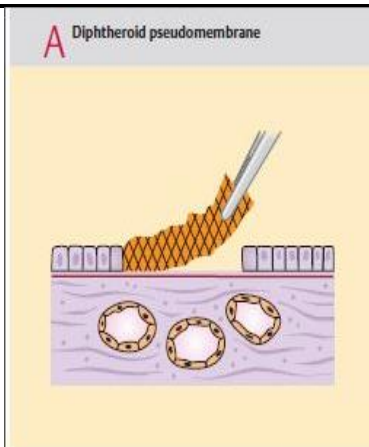


4. Catarrhal inflammation:

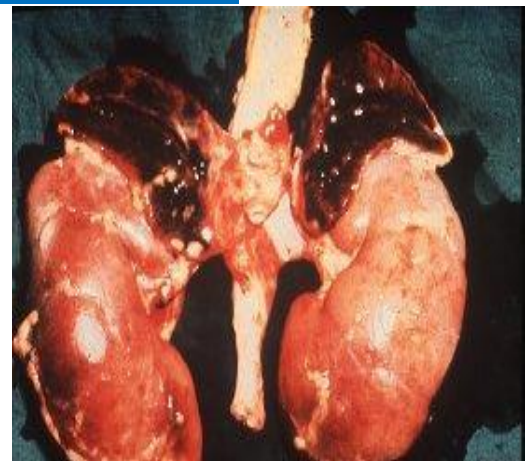


Acute laryngitis with petechiae
(from Giarelli L. 1987)

5.Pseudomembranous inflammation (diphtheria):



6. Hemorrhagic inflammation of adrenal cortex:



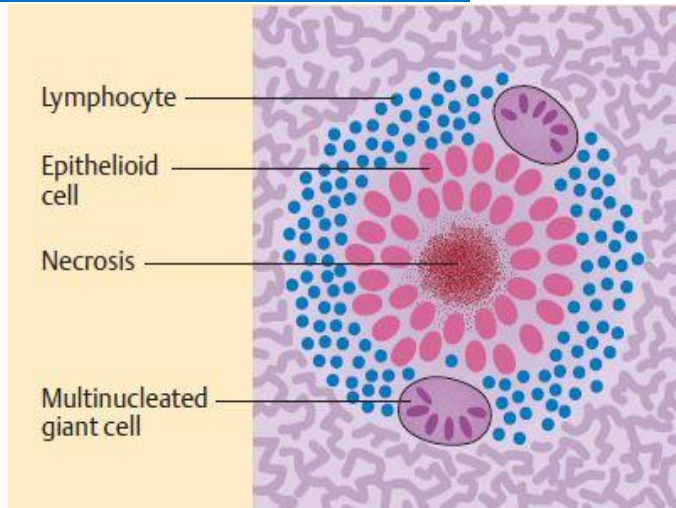
7.Necrotizing inflammation (cancrum oris):



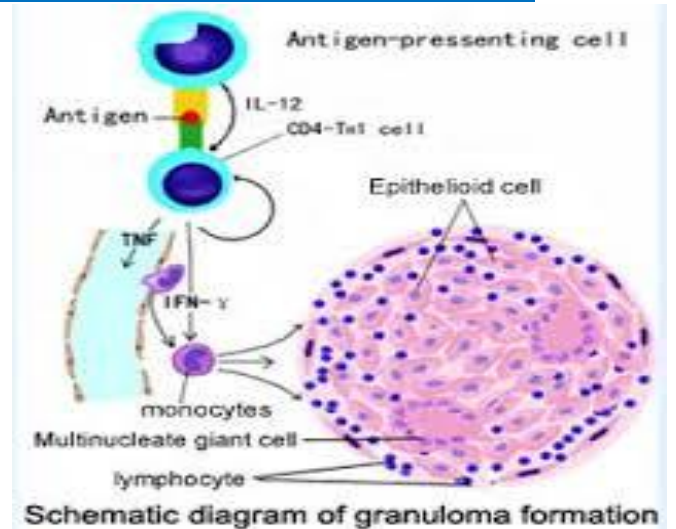
8.allergic inflammation (urticarial):



Granuloma: microscopic picture



Mechanism of granuloma formation:



Cells of inflammation

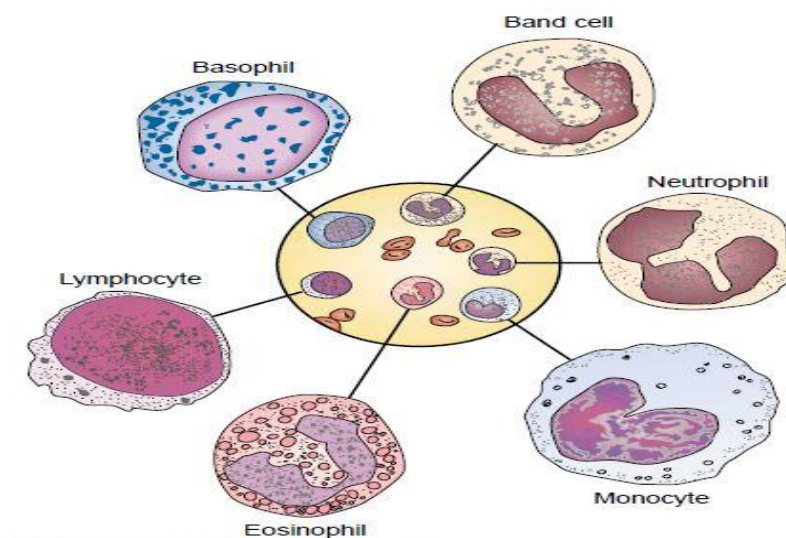
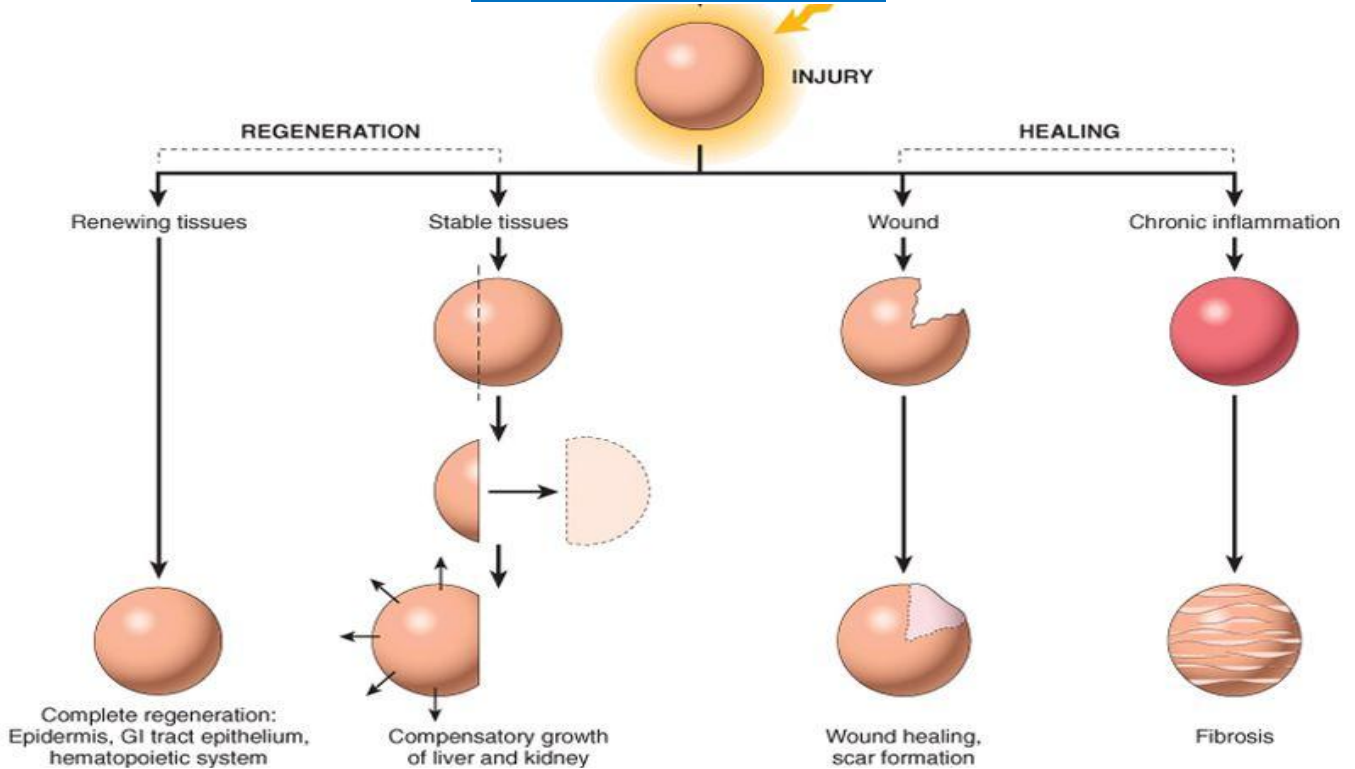


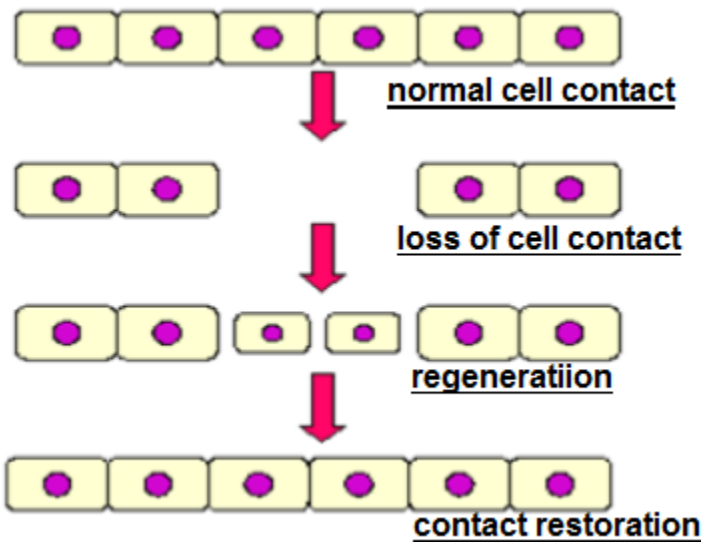
FIGURE 9-2 White blood cells.

Repair

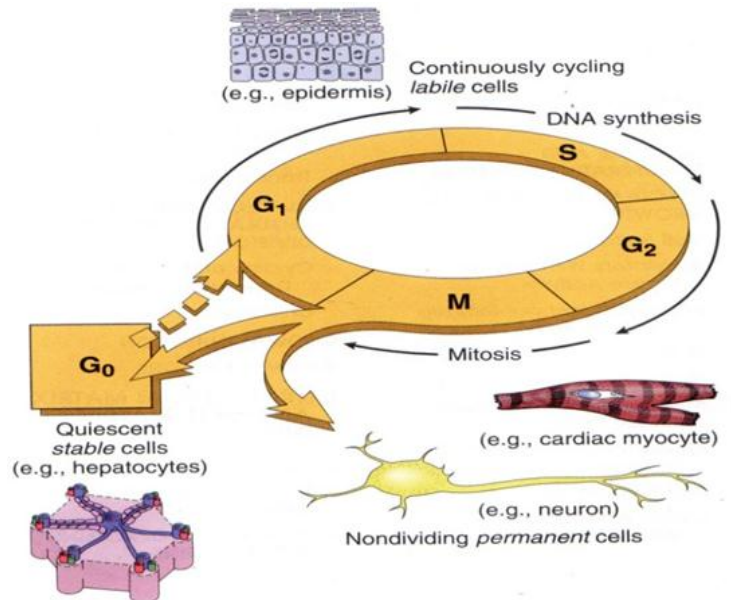
Response of body to injury



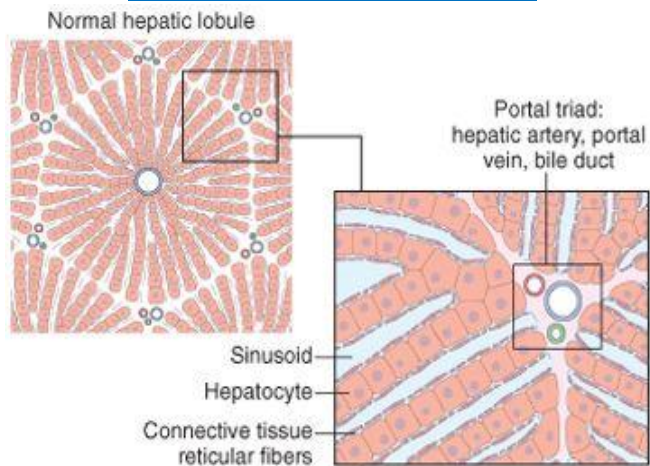
Contact inhibition



Cell cycle and proliferative capacity of cells

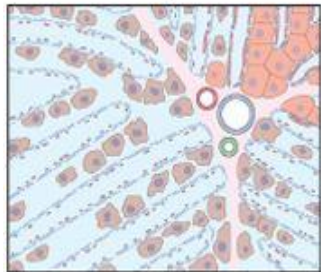


Regeneration of liver cells

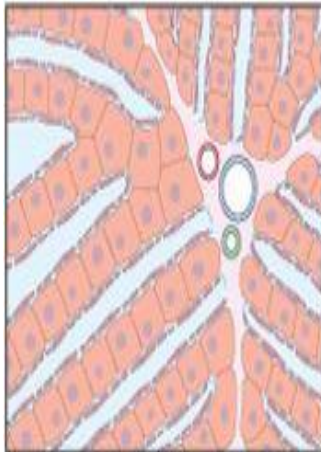


Injury to cells

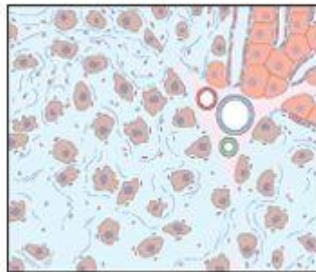
Injury to cells and matrix



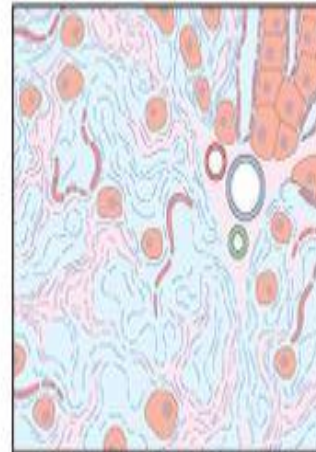
Proliferation of residual cells within intact matrix



REGENERATION



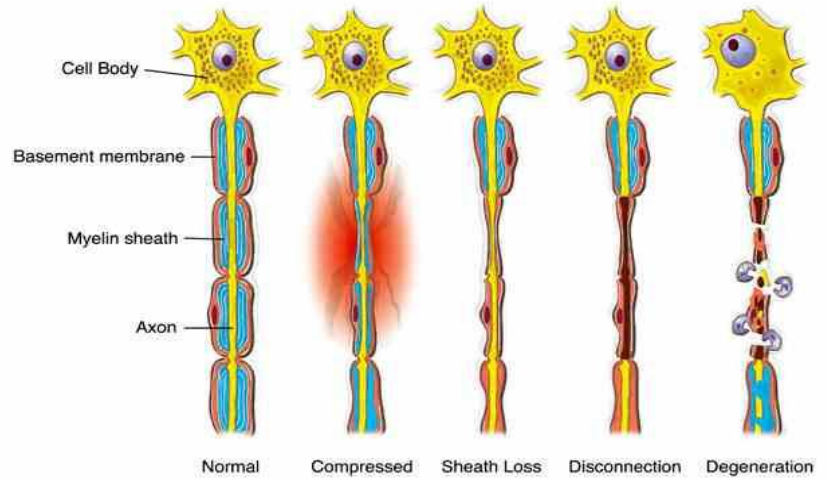
Deposition of connective tissue; proliferation of residual cells within disrupted matrix



REPAIR BY SCARRING

Healing of nervous system

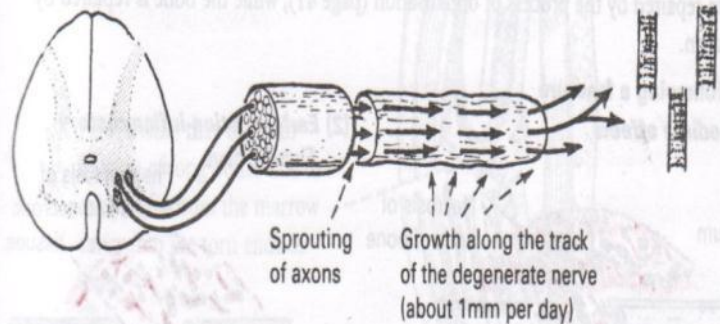
a. results of nerve injury:



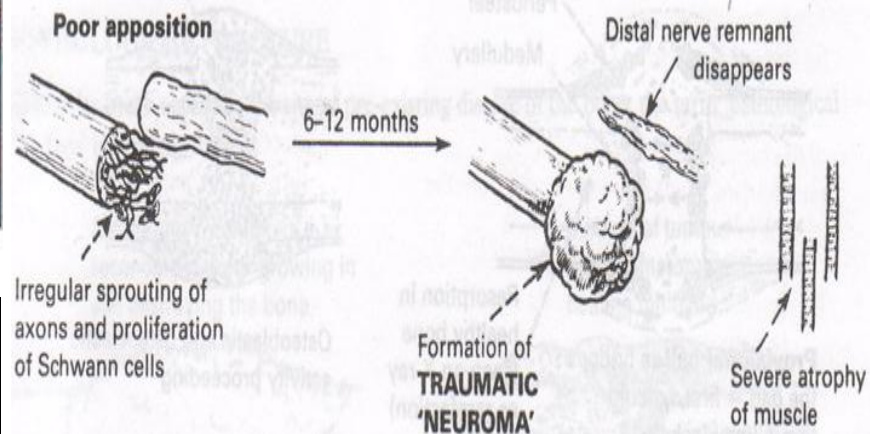
b. healing of peripheral nerve:

PERIPHERAL NERVES (continued)

Regeneration takes the form of a sprouting of the cut ends of the axons.

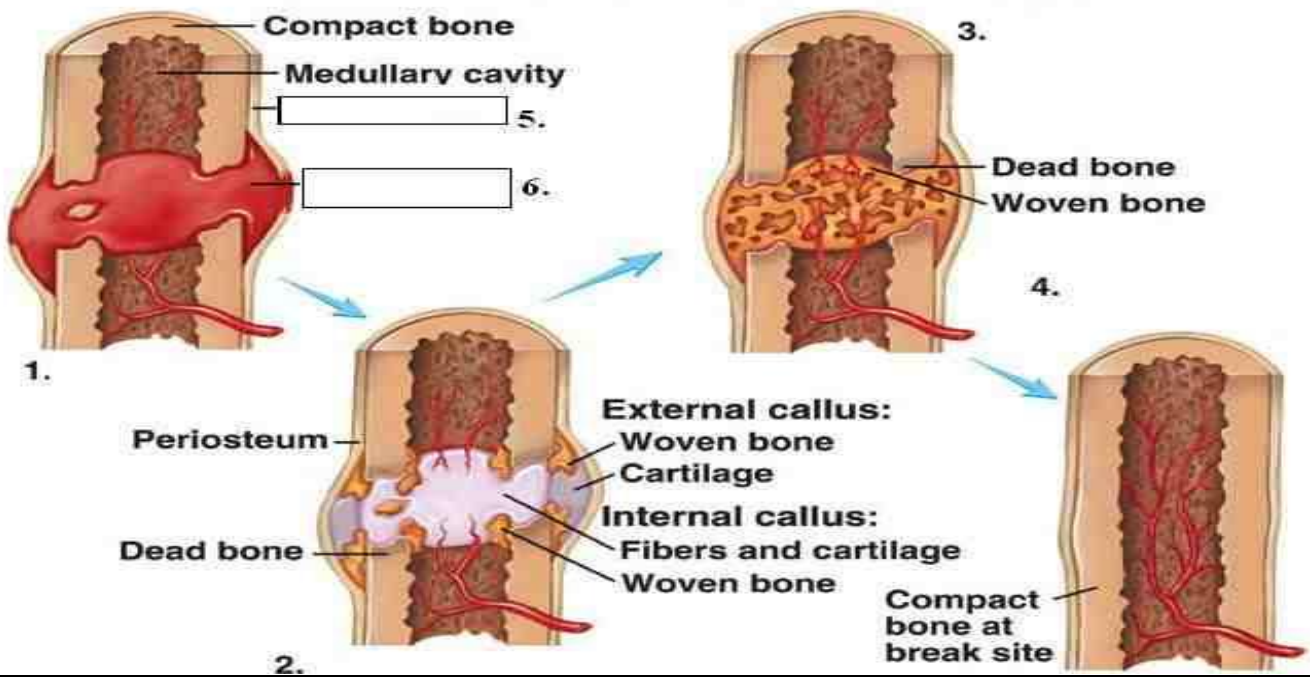
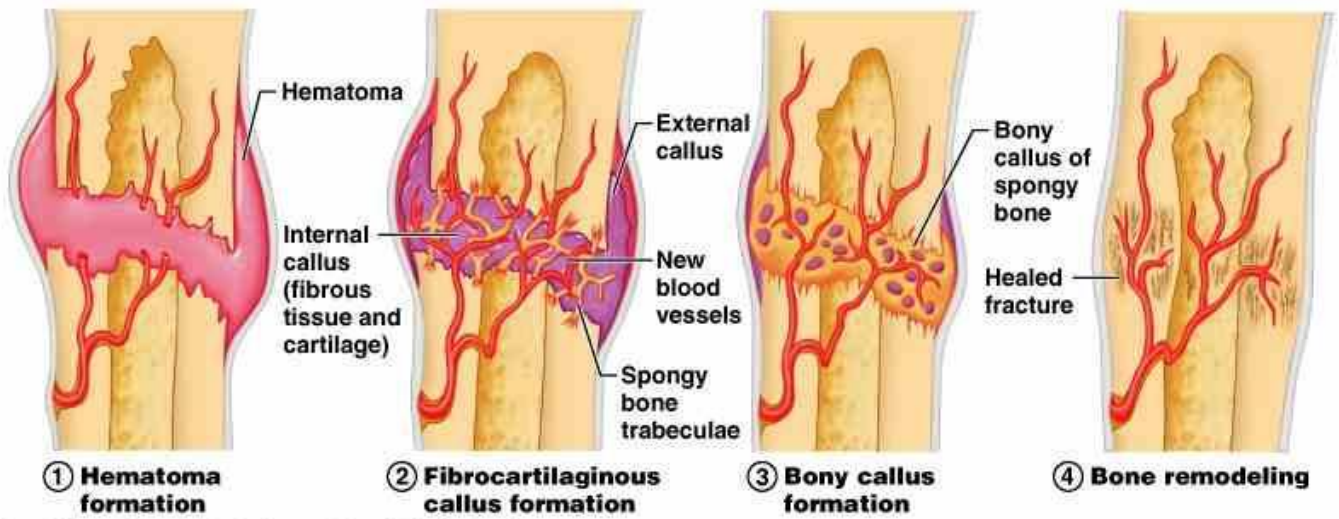


c. traumatic neuroma

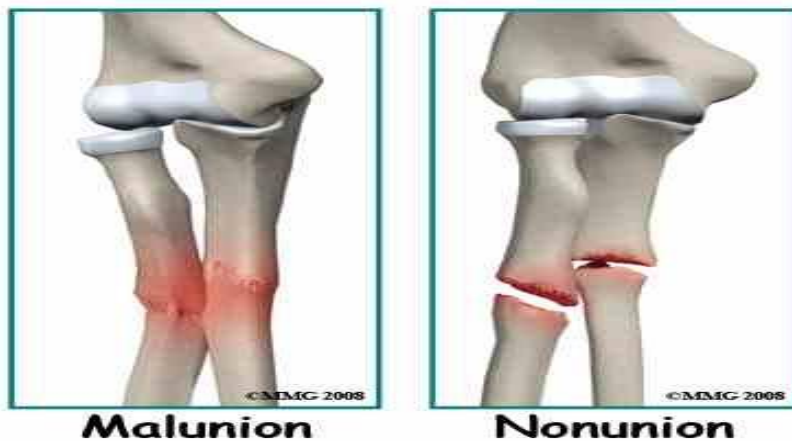


Regeneration of bone fracture

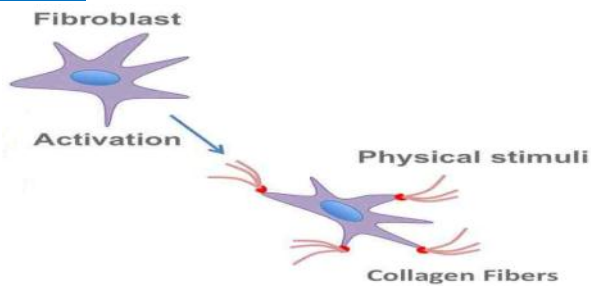
a. mechanism of regeneration:



b. complication:

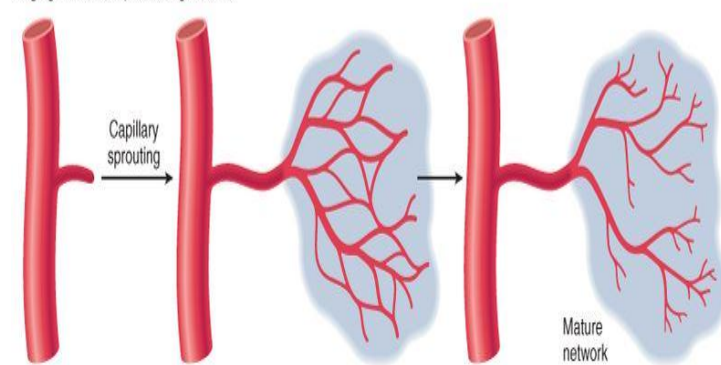


Fibroblast:

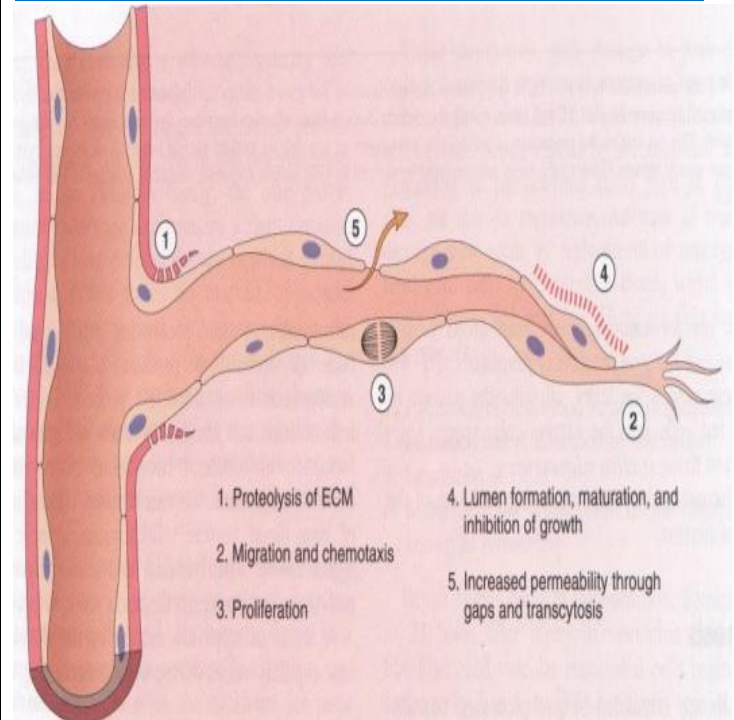


Angiogenesis:

Angiogenesis from preexisting vessels

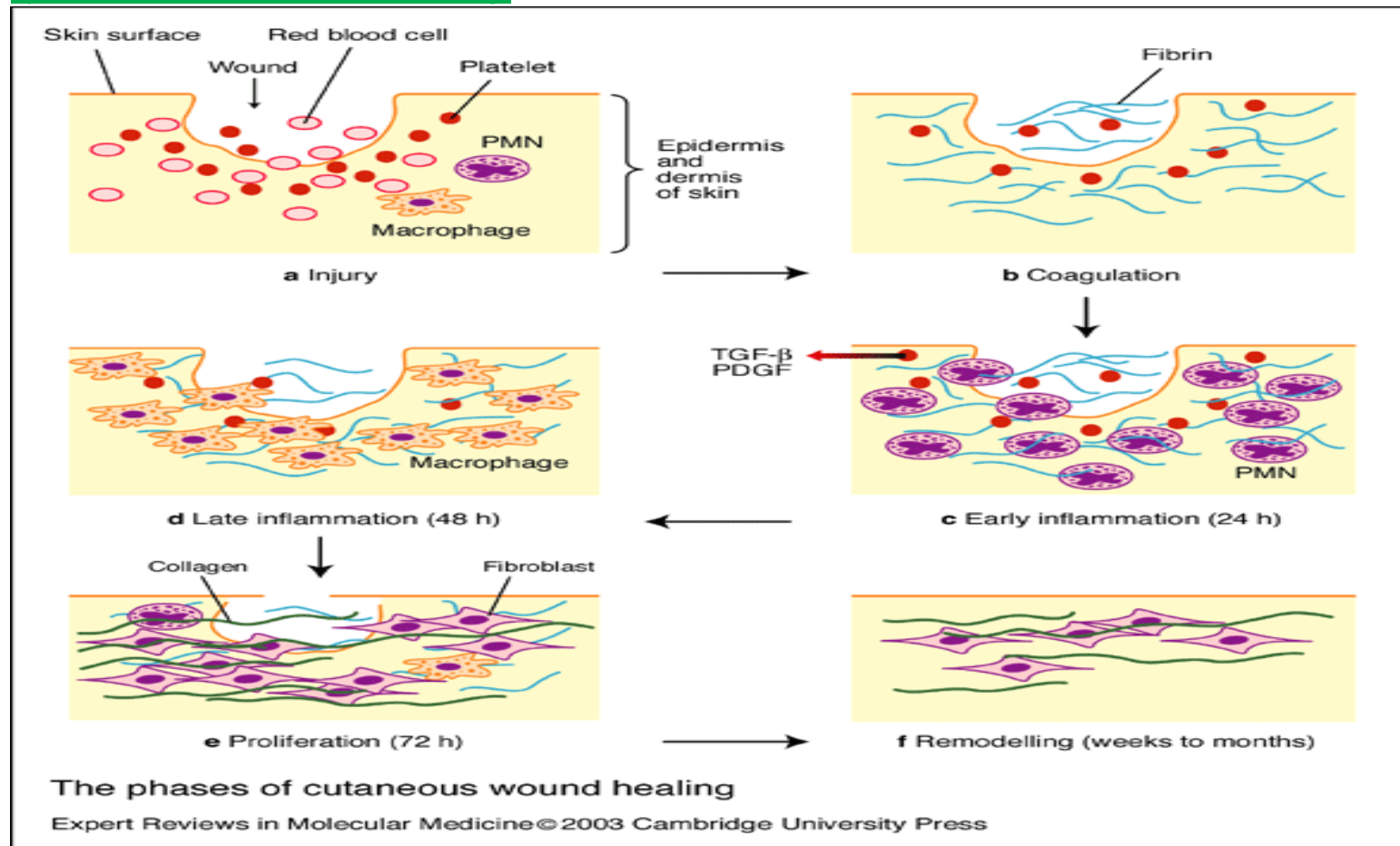


Mechanism of granulation tissue formation:

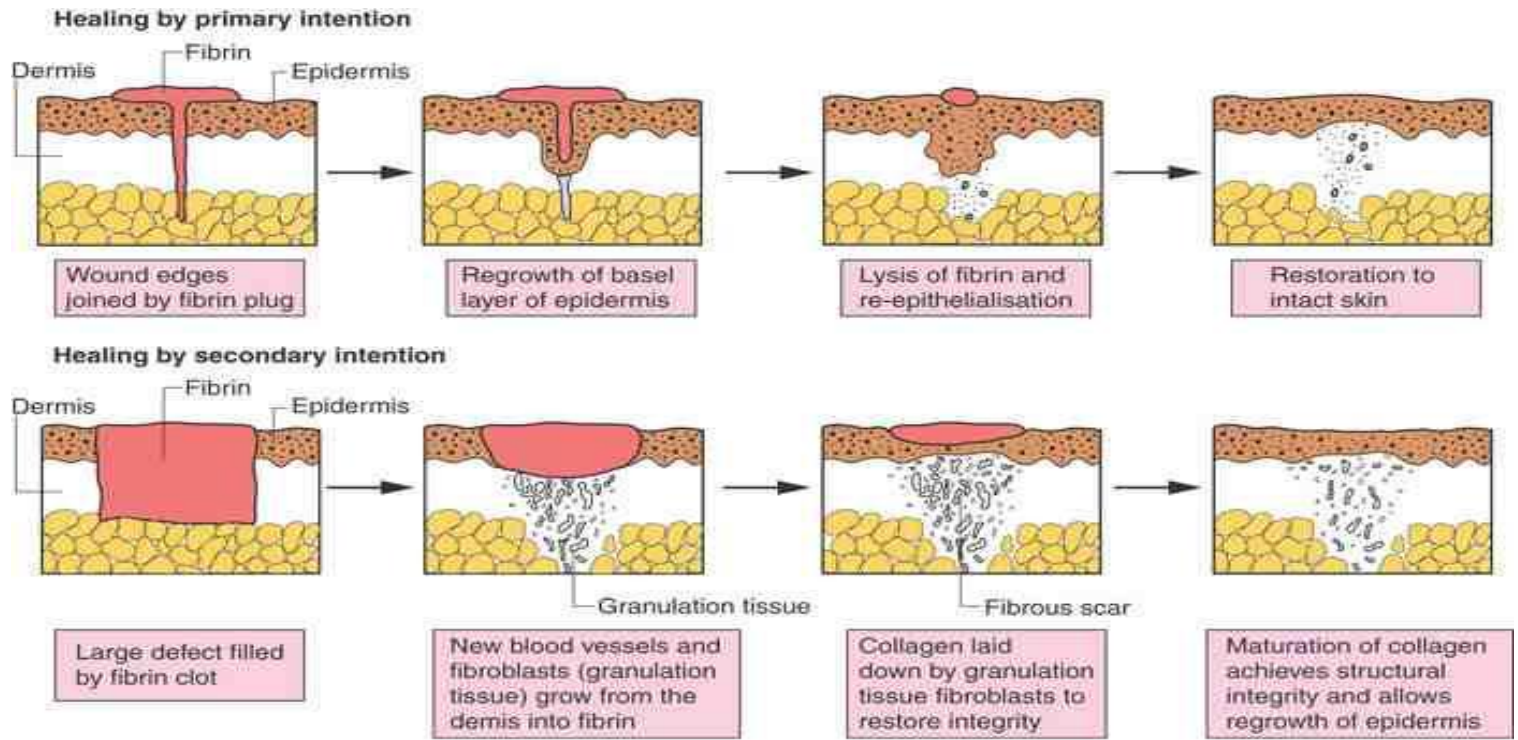


wound healing

*phases of continuous wound healing:



*types of wound healing:

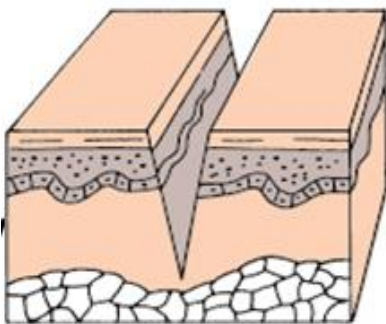


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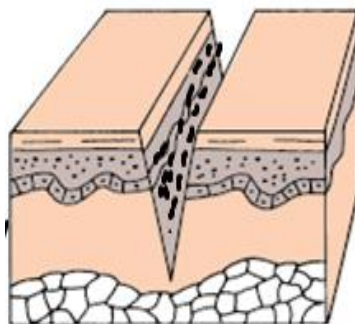
Characters of wound healing

Characters of wound healed by 1st intention

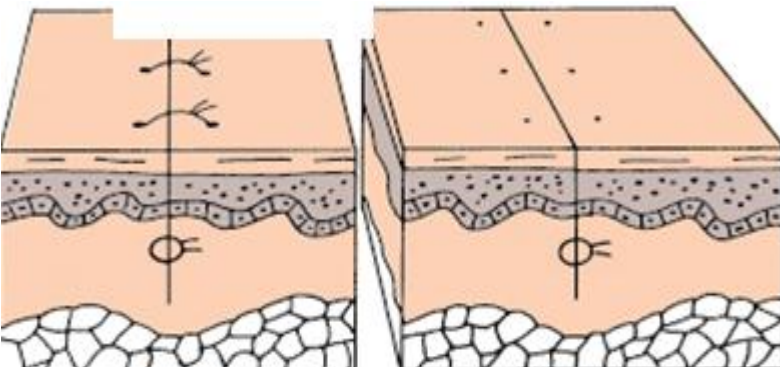
1. clean, no gap and minimal tissue loss:



2. minimal granulation tissue:

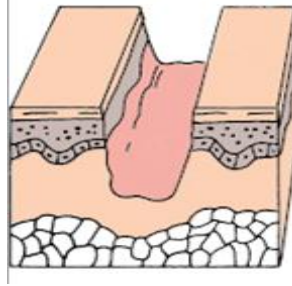


3. no wound contraction (thin scar)

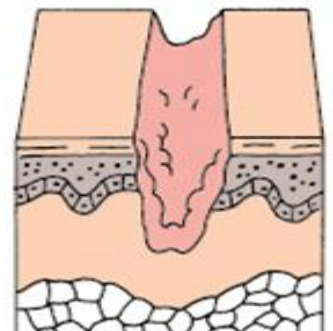


Characters of wound healed by 2nd intention

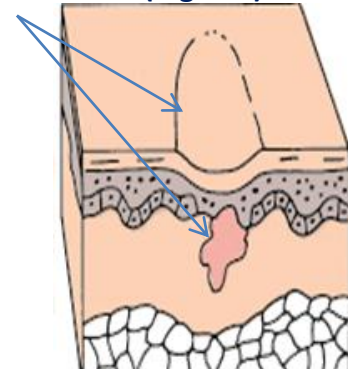
1. infected, wide wound gap and marked tissue loss:



2. excessive granulation tissue:

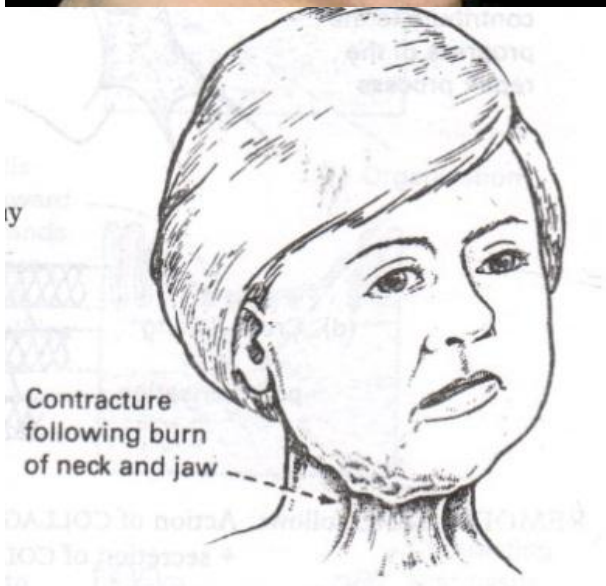


3. wound contraction (big scar)



Complications of wound healing

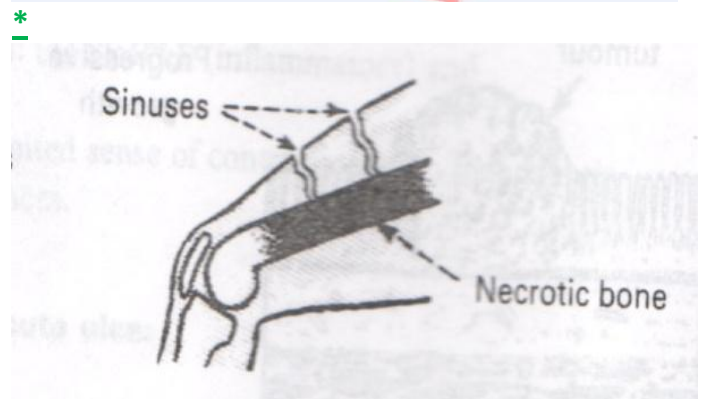
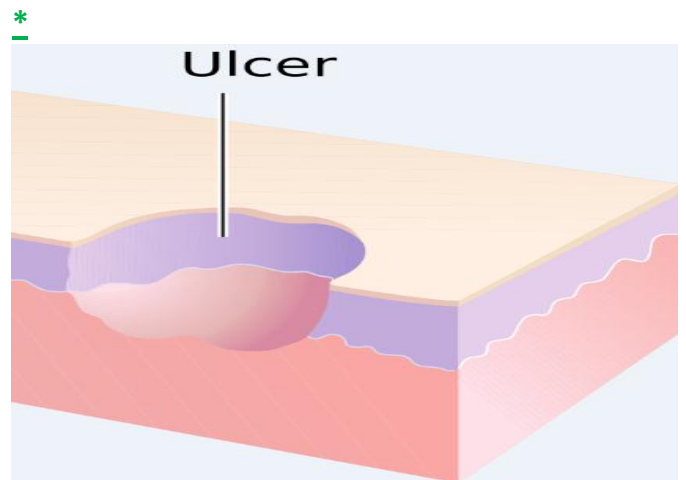
1.cosmotic deformity



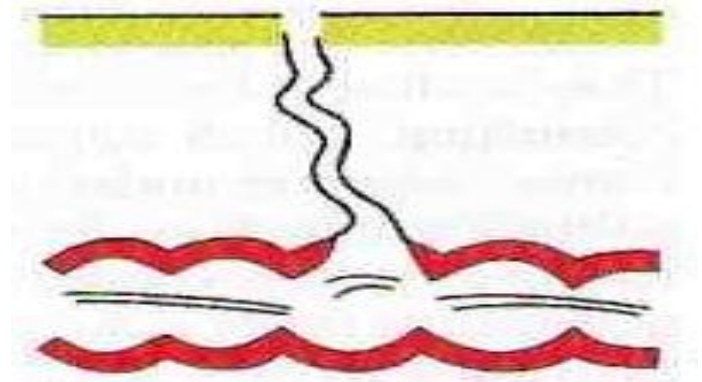
3.keloid



2.chronic ulcer,sinus and fistula



*Fistula



4.functional trouble (wound contracture around joint of hand)



5.epidermoid cyst

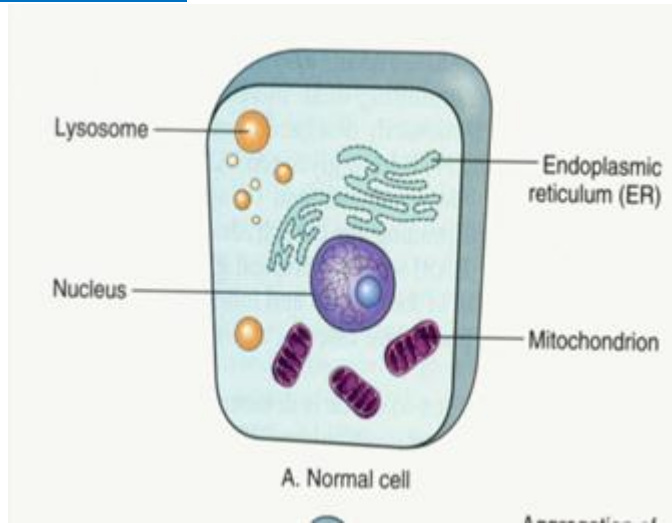


6.carcinoma (Marjolin's ulcer)

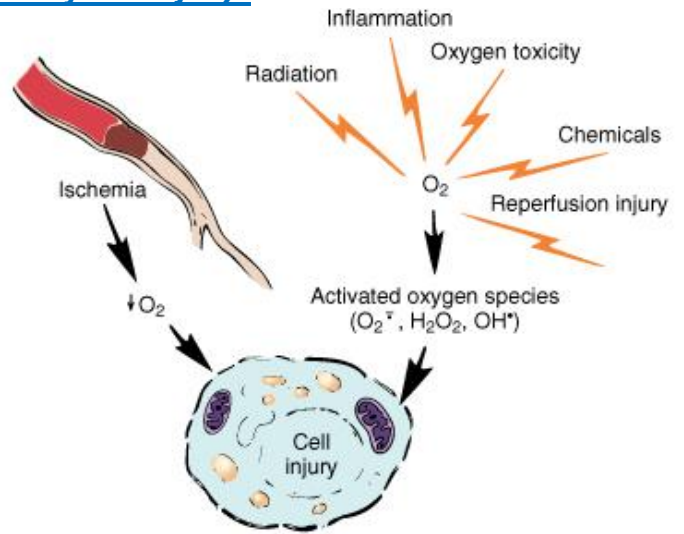


Cell injury

Normal cell:

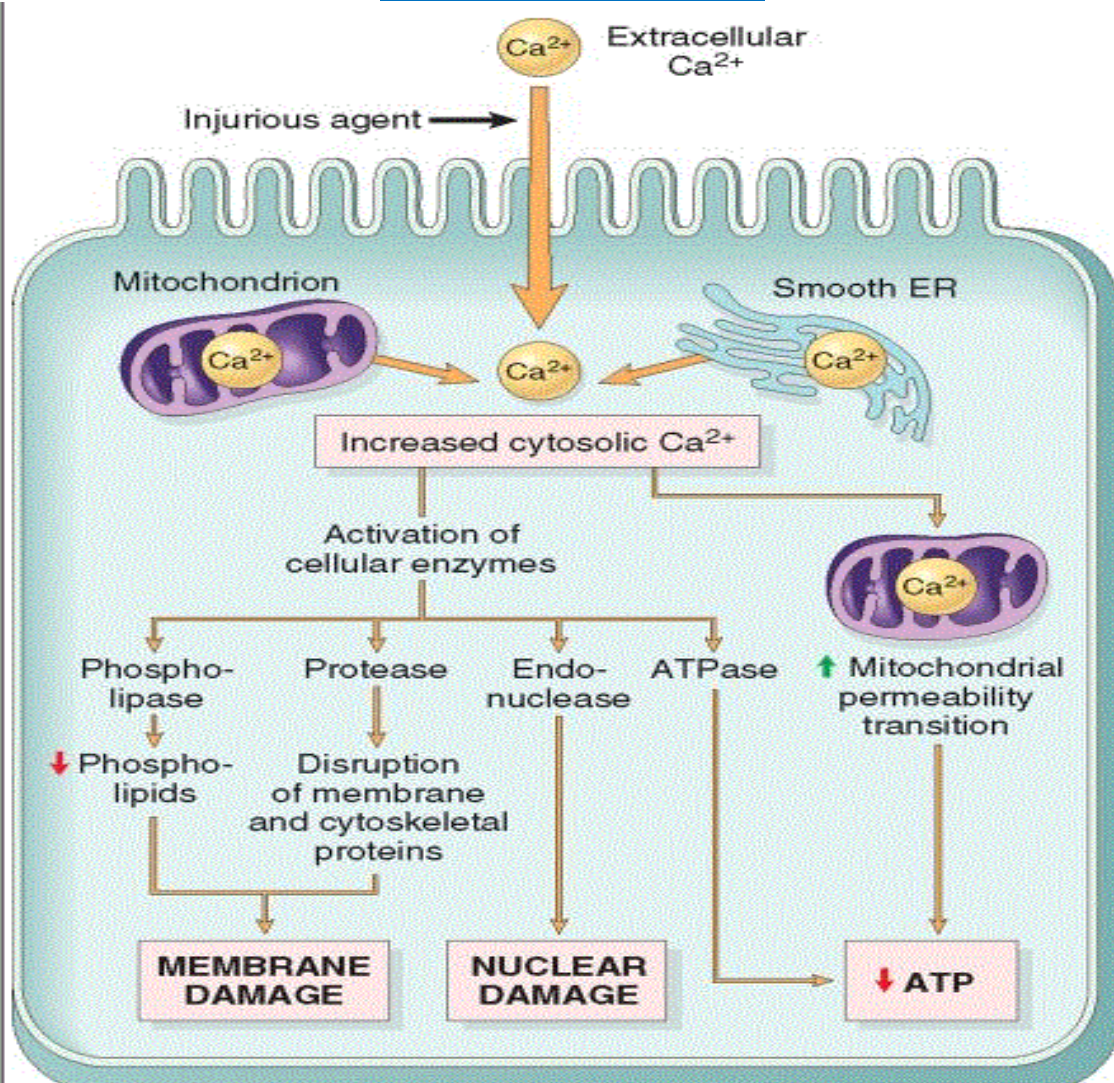


Causes of cell injury:

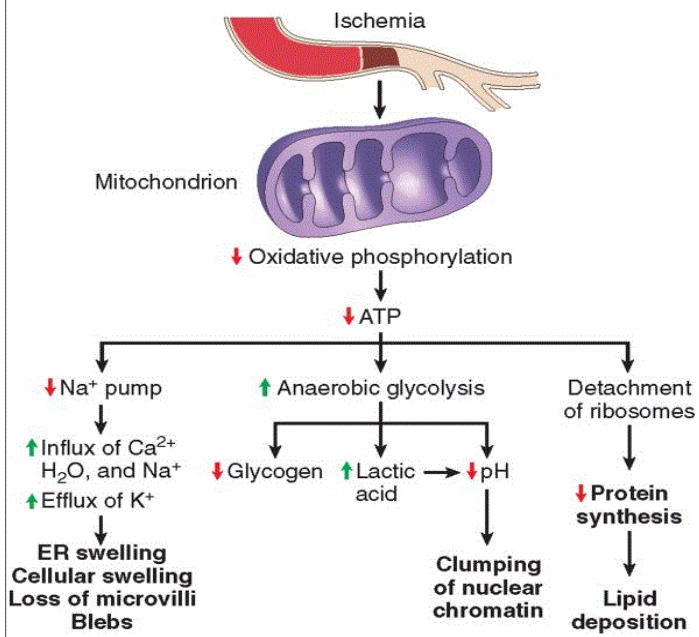


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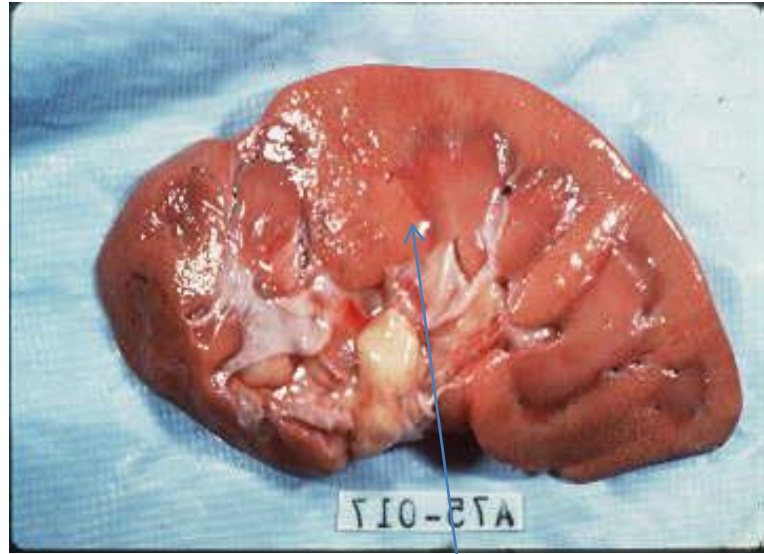
Mechanism of cell injury



Pathogenesis of cell swelling:

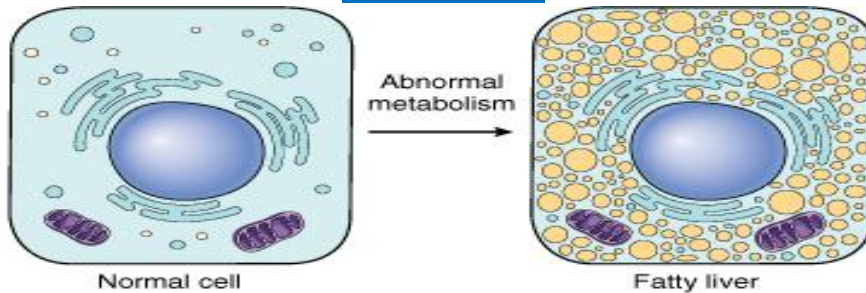


Hydropic change of the kidney:

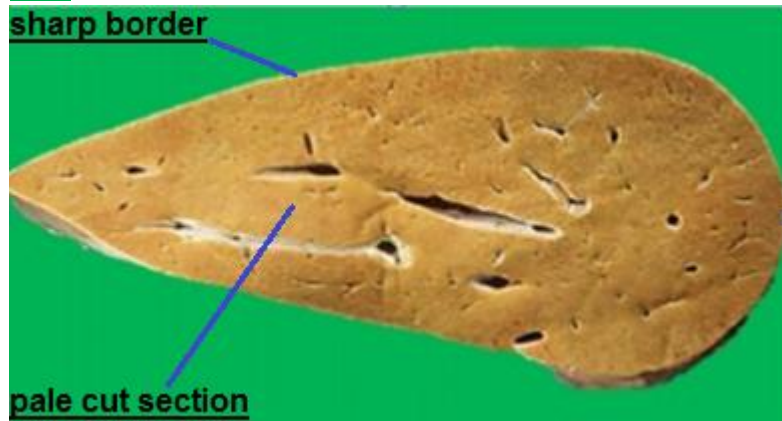


pale kidney due to ischemic changes

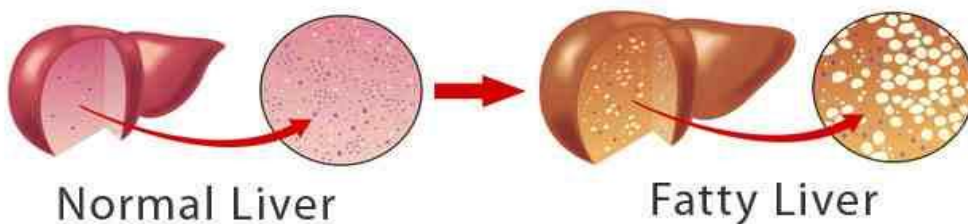
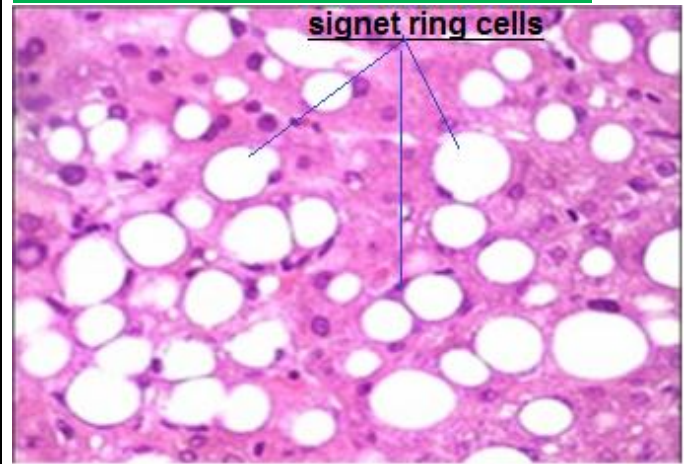
Fatty change



Gross picture of hepatic steatosis: pale cut section of liver

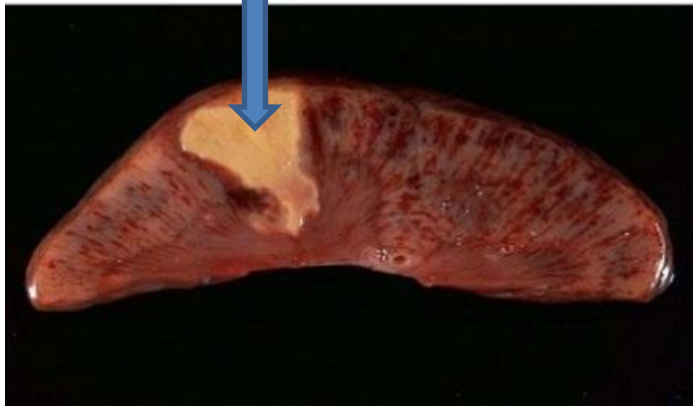


Microscopic picture of hepatic steatosis:



coagulative necrosis:

Kidney Infarct - coagulative necrosis



caseous necrosis (pulmonary tuberculosis):

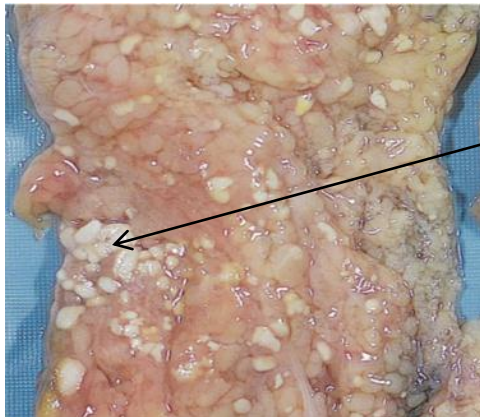


gangrenous necrosis (hand):



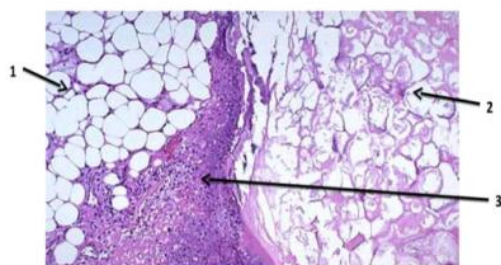
Fat necrosis:

a.gross picture: foci of fat necrosis in the mesenteric:



B. microscopic picture:

Fat Necrosis

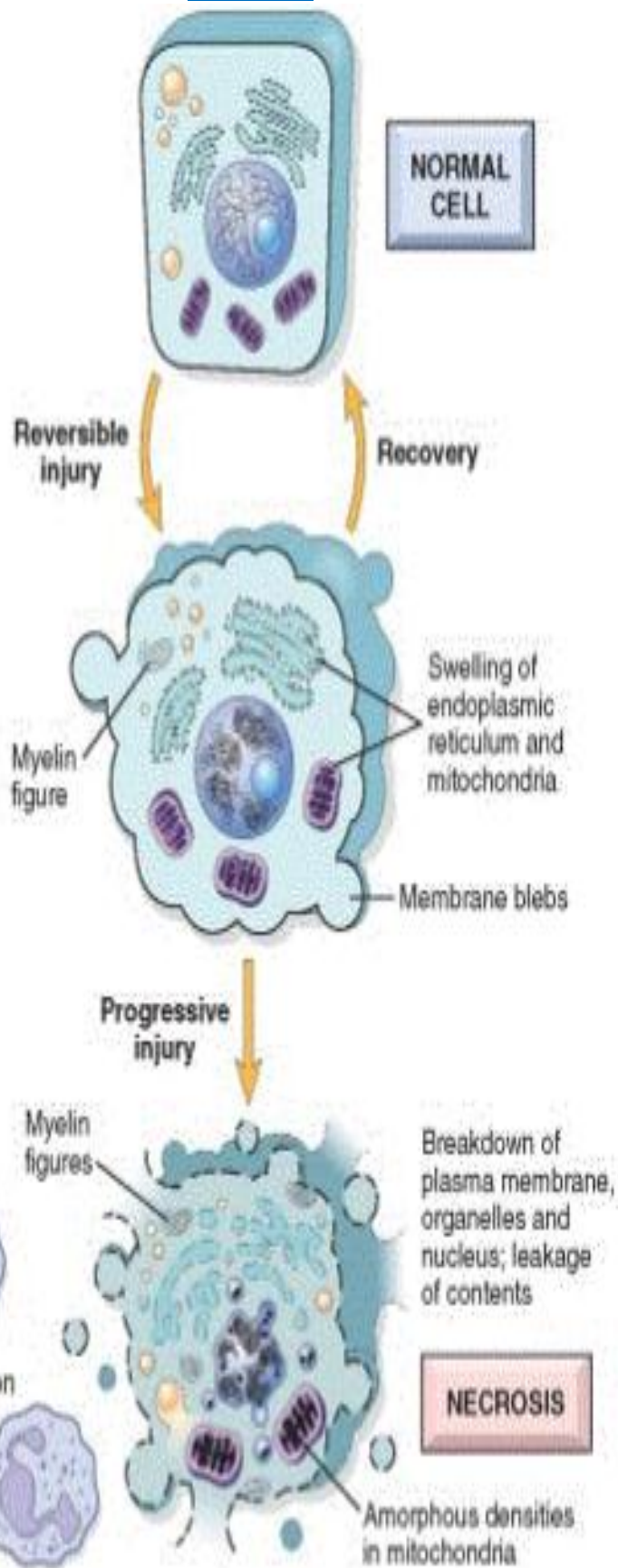


- 1) Lobule of normal-looking fat cells
- 2) Lobule of necrotic fat cells forming cystic spaces
- 3) Inflammatory cells

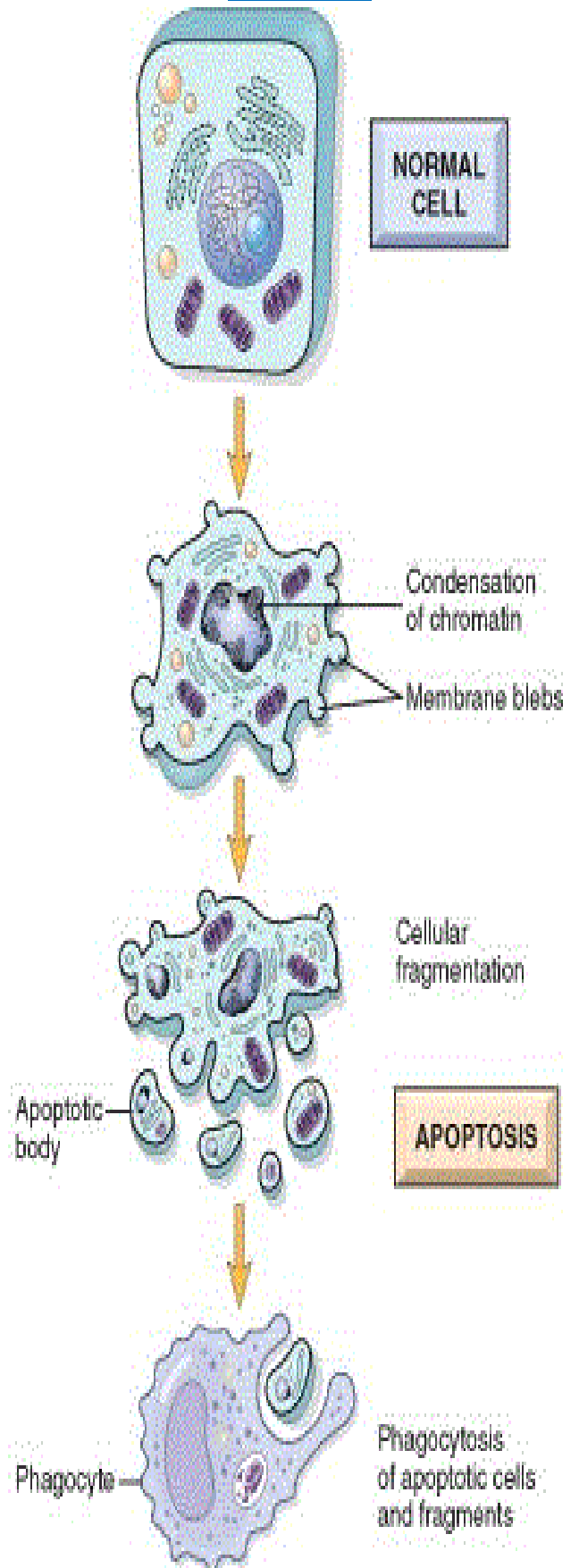
Liquifactive necrosis



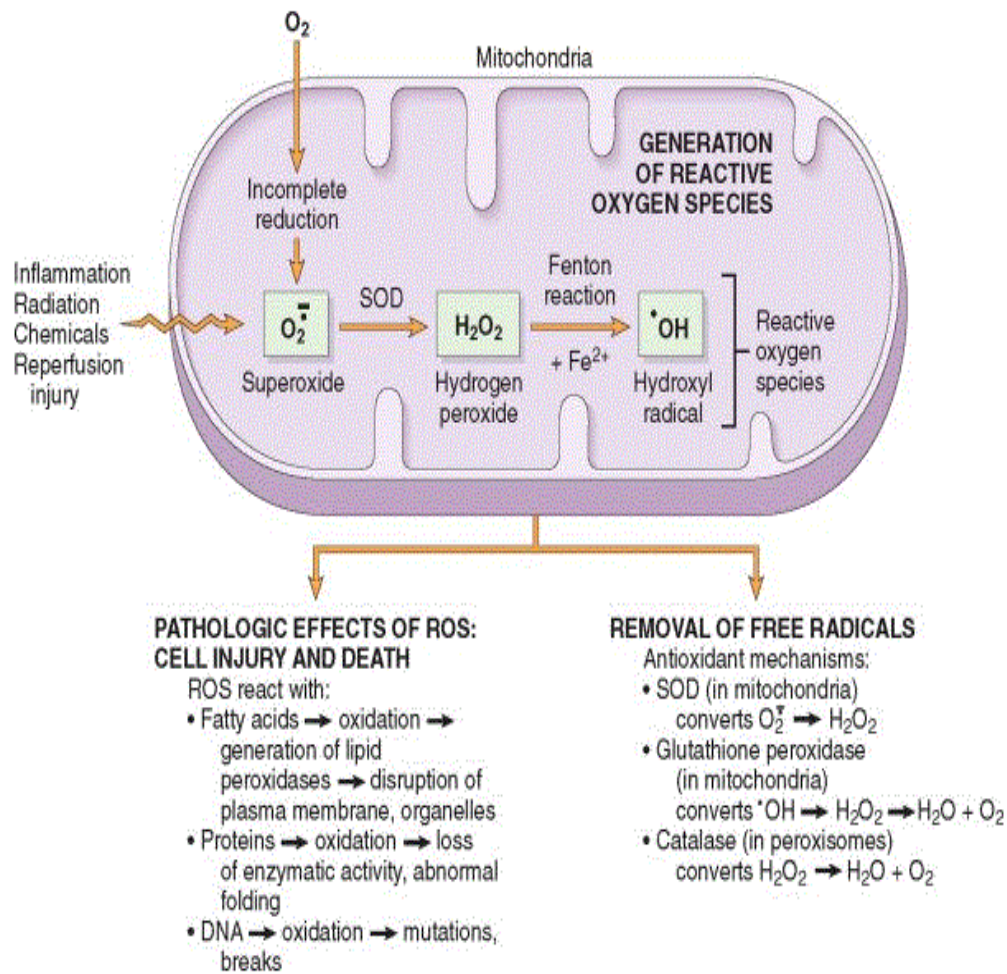
Necrosis



Apoptosis

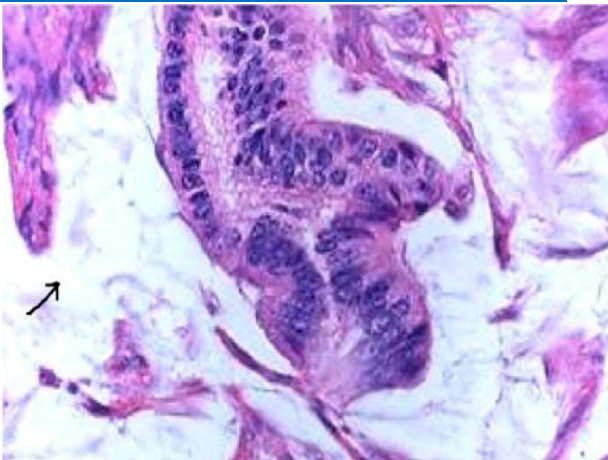


Free radical

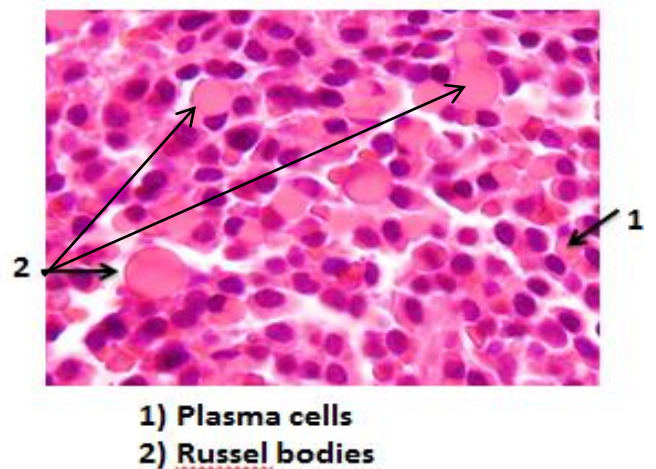


Cellular accumulation

Mucoid degeneration (pale mucin pools)



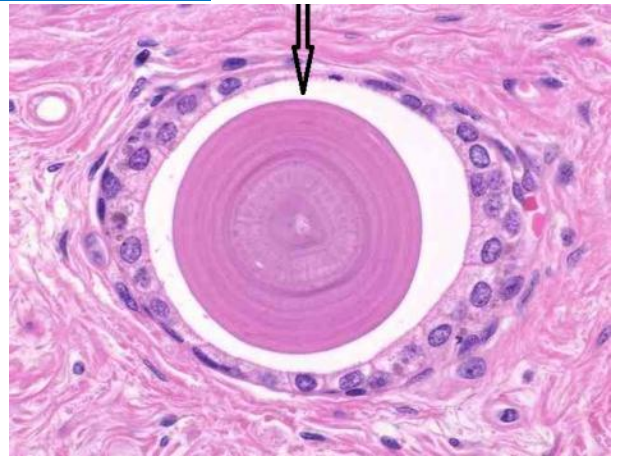
Russell bodies:



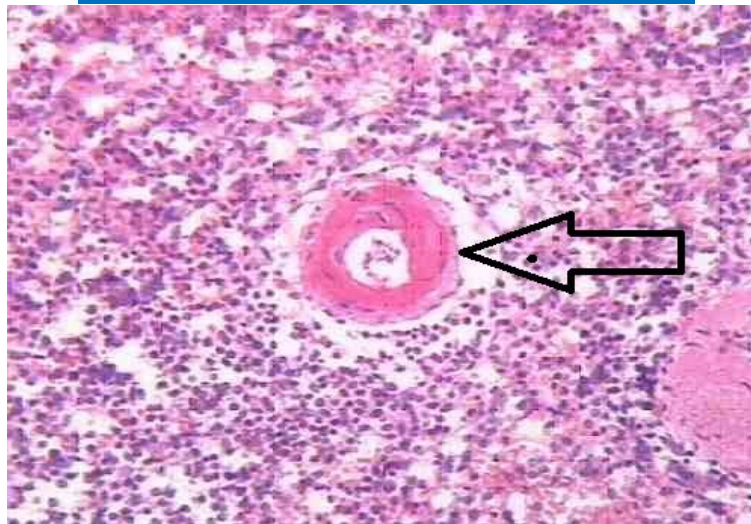
Mallory bodies



Corpora amylacea



Hyalinosis of arteriole in hypertension



Anthraxosis



Healthy lung



Early stages



Late stages

cafe'au lait skin patches



Tattooing



Albinism



Plumbism



Leukoderma

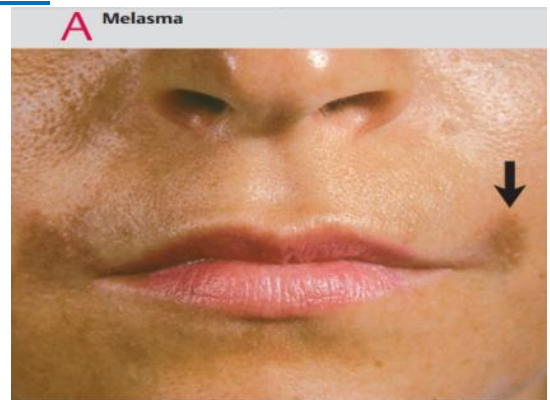


Skin affection in Addison's disease



Hyperpigmentation in Addison's Disease

Melasma



Brown atrophy of the heart

Gross picture:

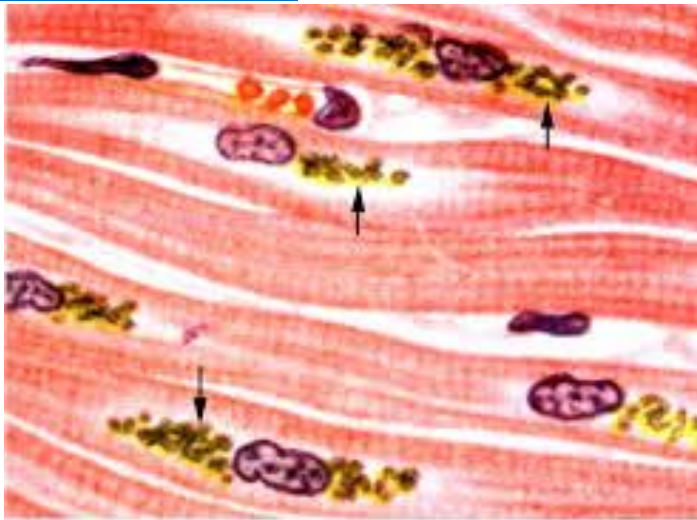


Melanosis coli

Gross picture:

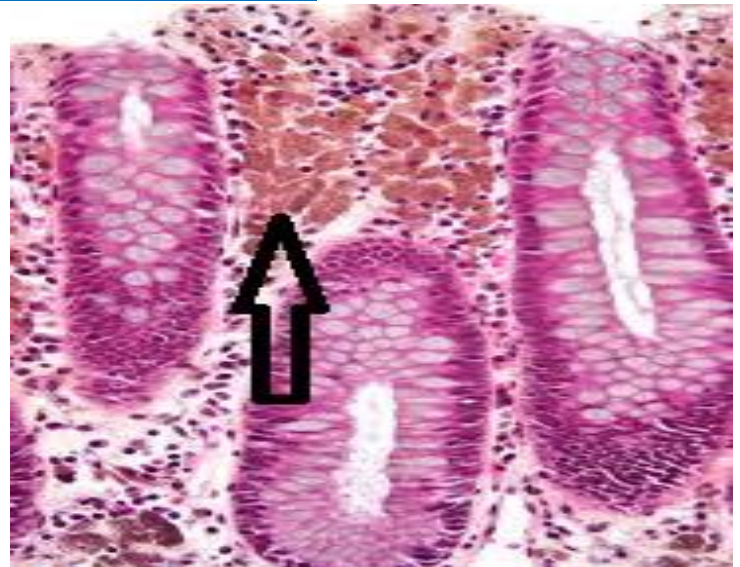


Microscopic picture:



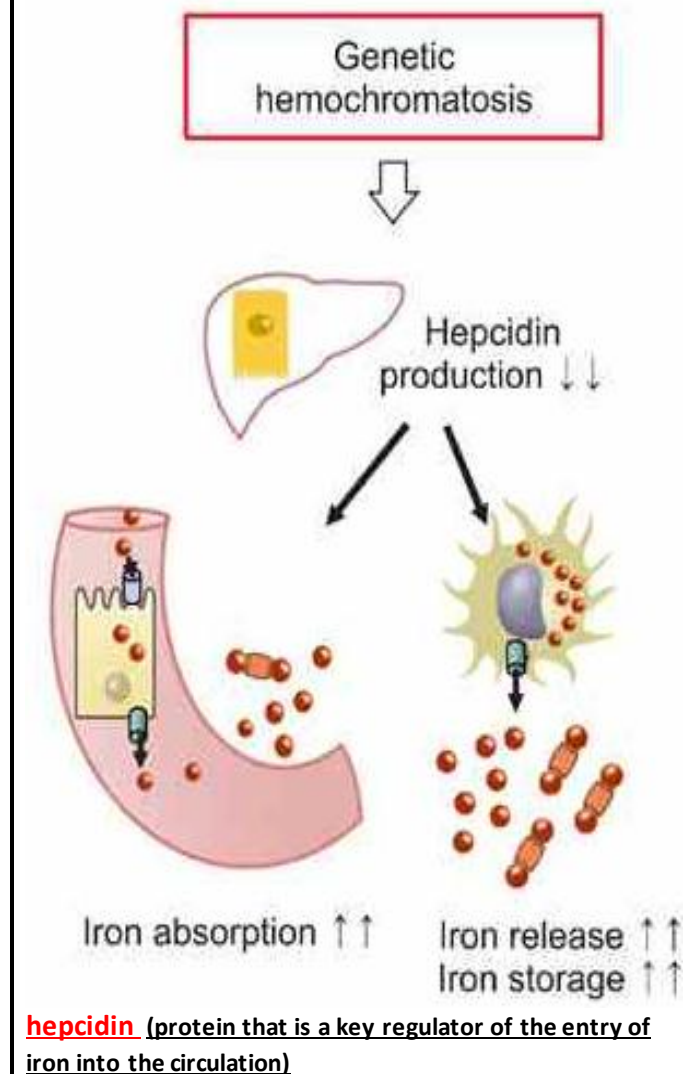
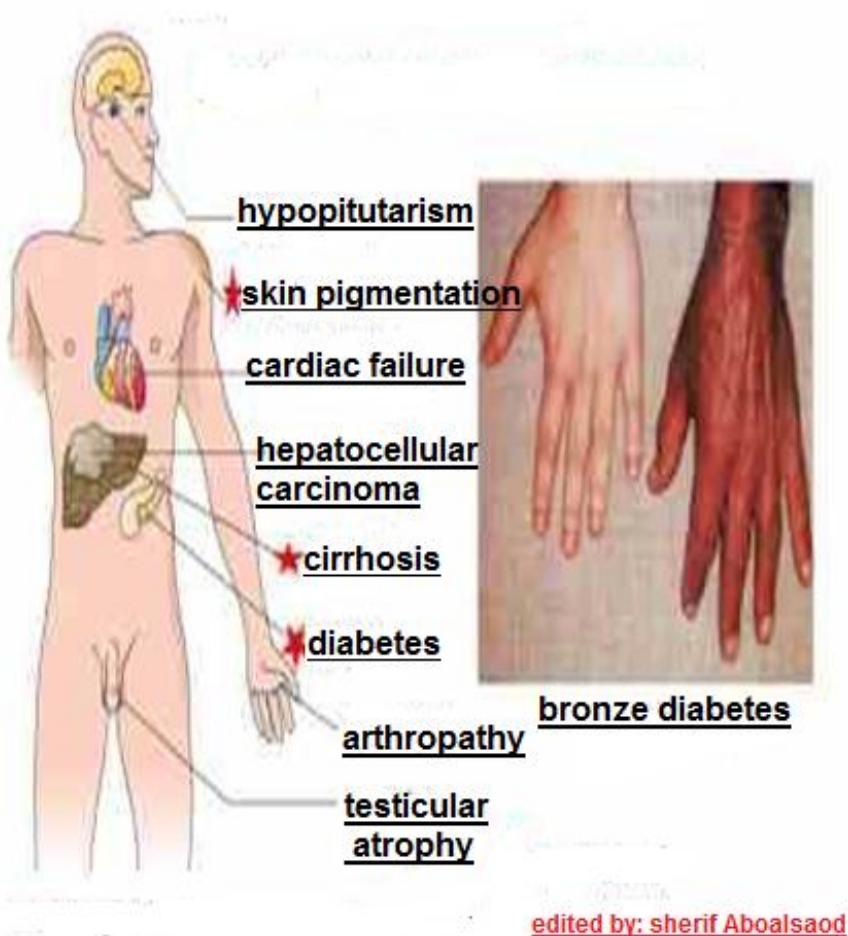
lipofuscin in cardiac cells

Microscopic picture:



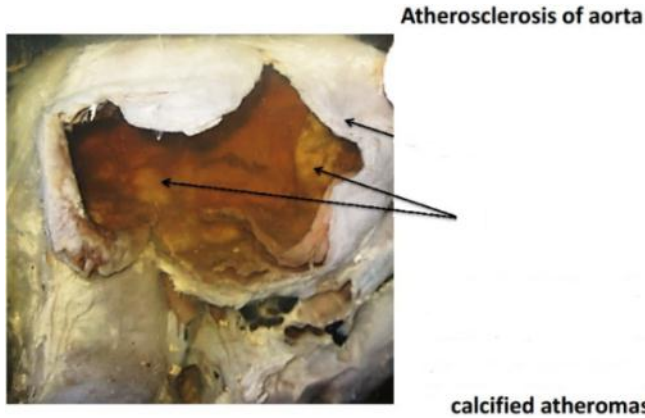
Lipofuscin between mucosal glands

Complication of hemochromatosis

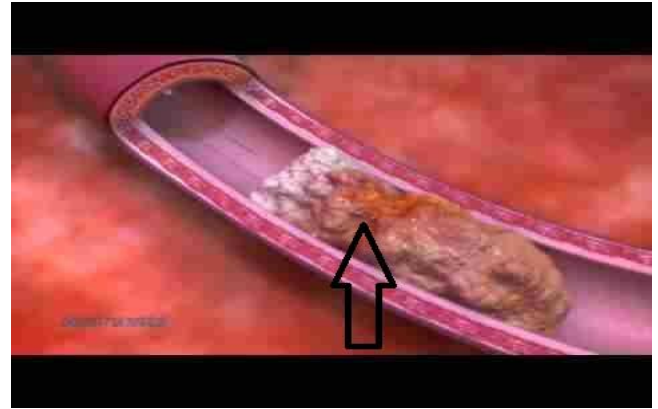


Pathological calcification

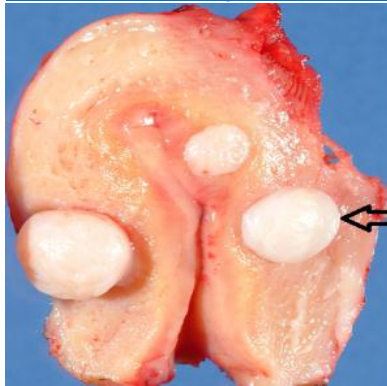
Atherosclerosis of aorta



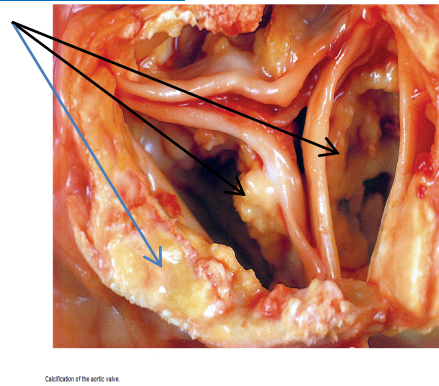
Calcified thrombus



Calcified Leiomyoma (uterus)



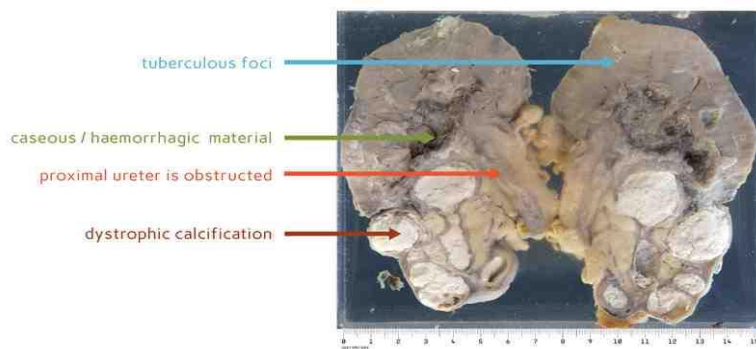
Calcified valve



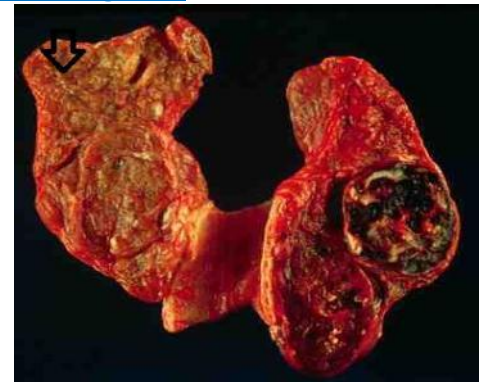
Lithopedion



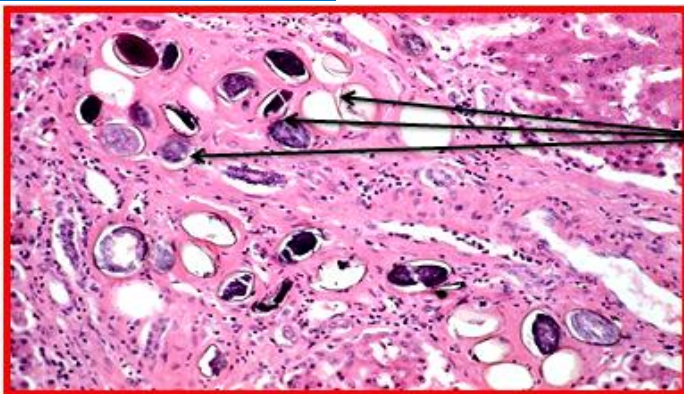
Calcified T.B lesion (kidney cut section)



Calcification in goiter

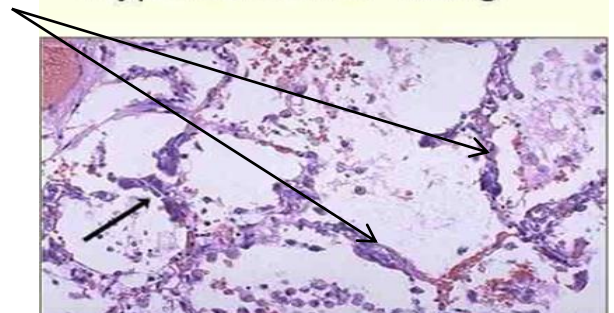


Calcification in Bilharziasis



Calcification in lung alveoli (blue granules in alveolar wall)

Metastatic Calcification
Hypercalcemia - Lung

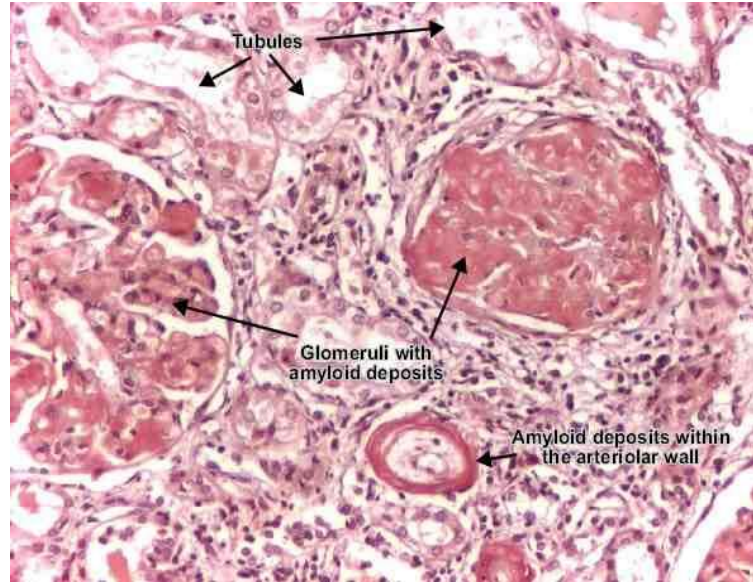


Kidney amyloidosis

Gross picture (pale gray waxy amyloid spot)



Microscopic picture

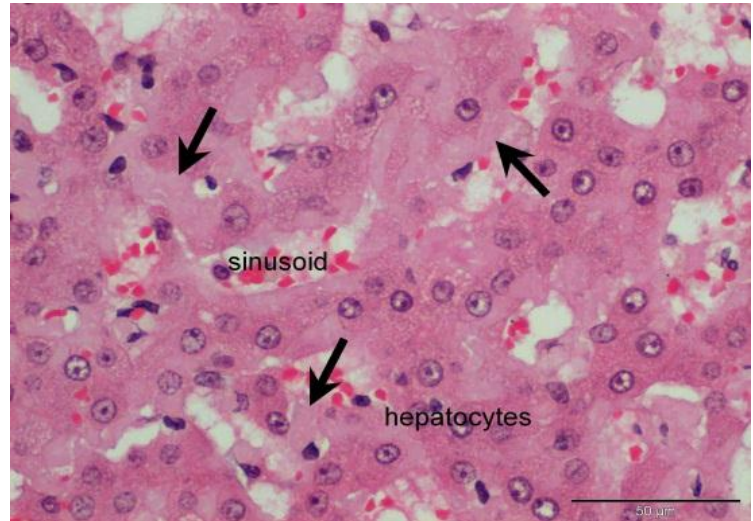


Liver amyloidosis

gross picture (pale gray waxy amyloid spot)



Microscopic picture



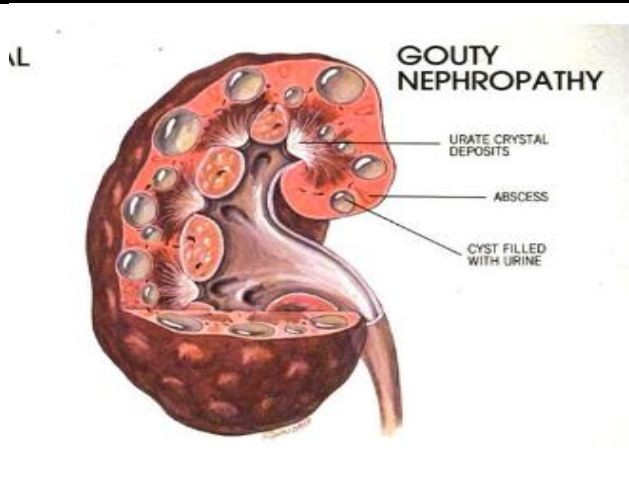
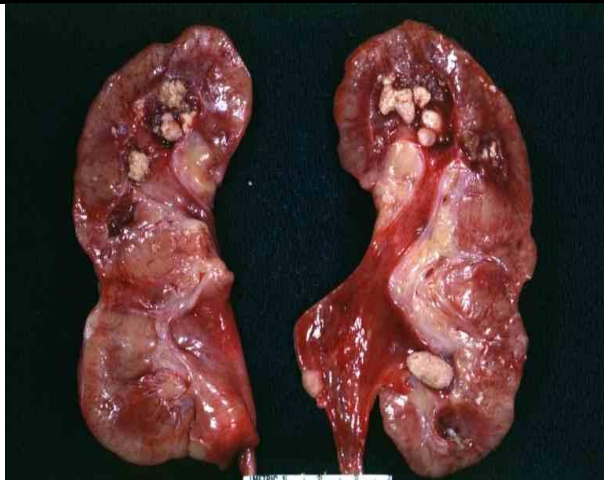
Spleen amyloidosis (cut section show pale amyloid spots)



Tophi



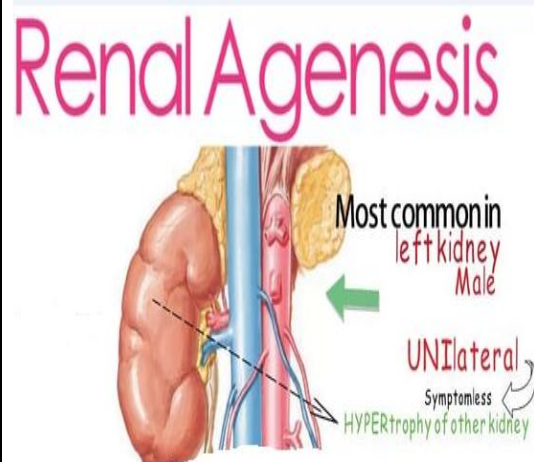
Gouty nephropathy



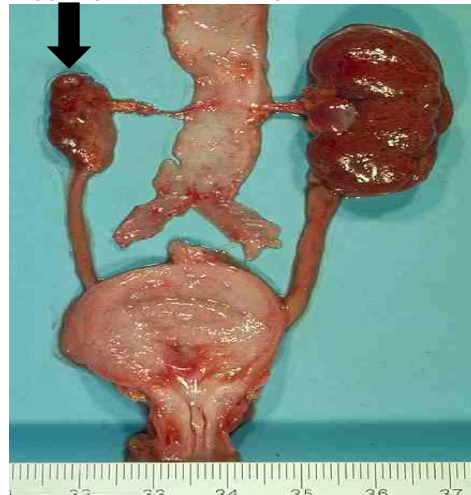
Growth disturbances

Congenital disorders

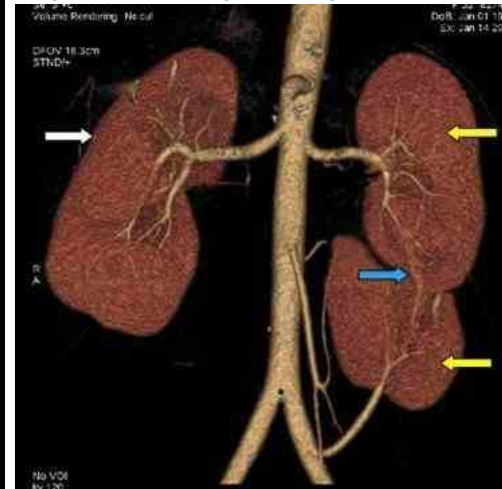
absent kidney



Hypo plastic kidney



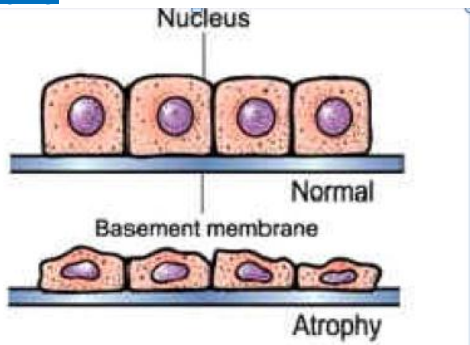
Supernumerary kidney:



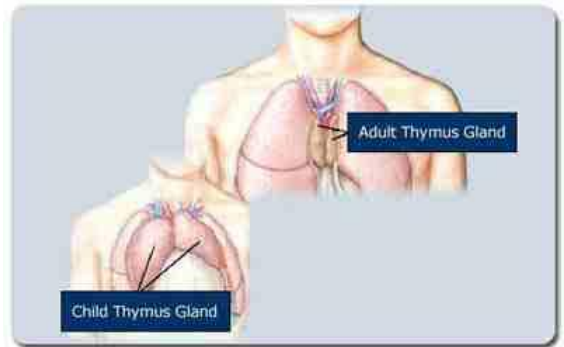
Acquired disorders

Atrophy:

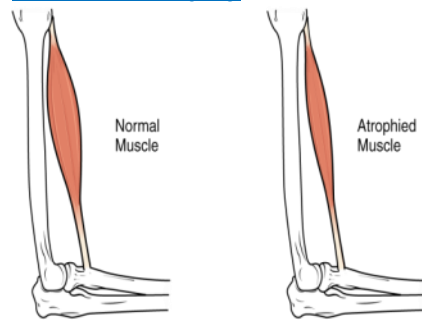
Cellular atrophy



Thymus atrophy

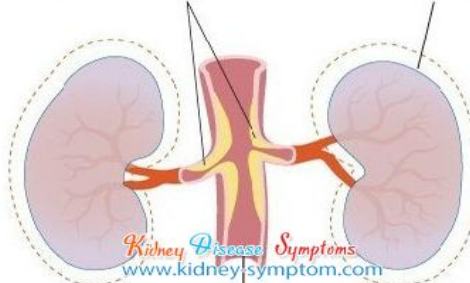


Muscle atrophy



Kidney atrophy

Atherosclerosis in aorta and renal arteries
Kidney size is reduced

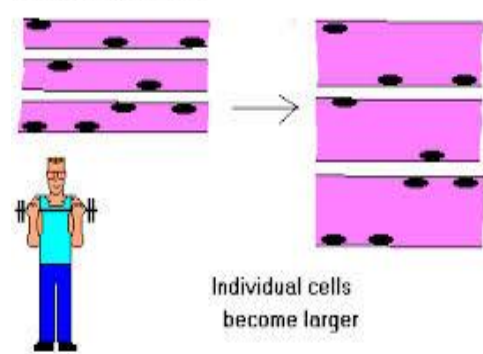


Progeria



Hypertrophy:

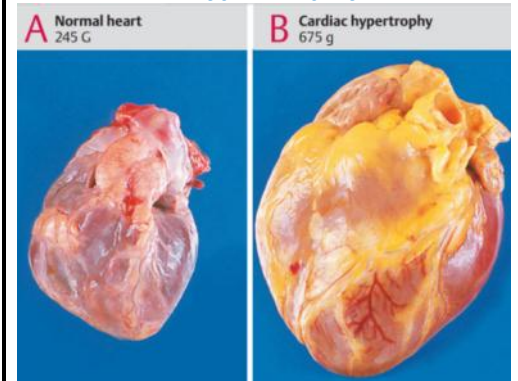
Hypertrophy



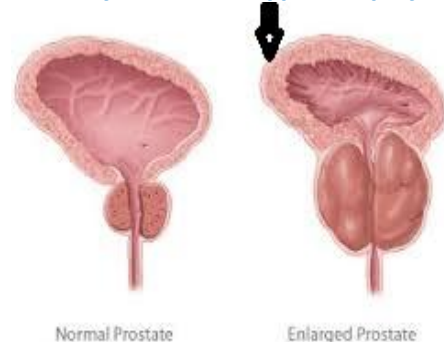
Muscle hypertrophy



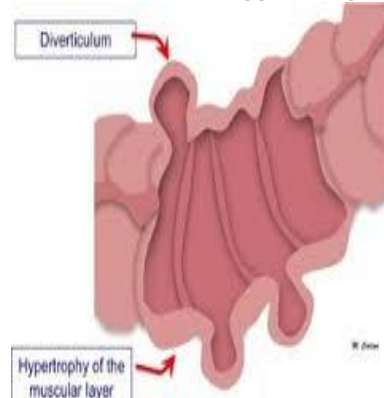
Ventricular hypertrophy



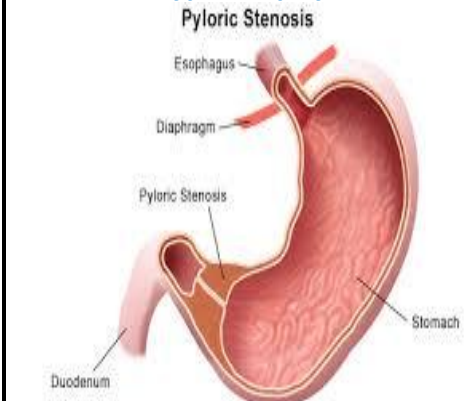
Urinary bladder hypertrophy



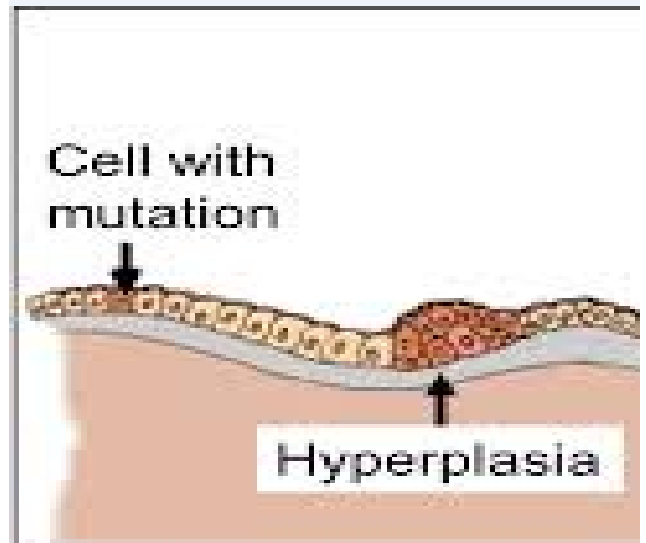
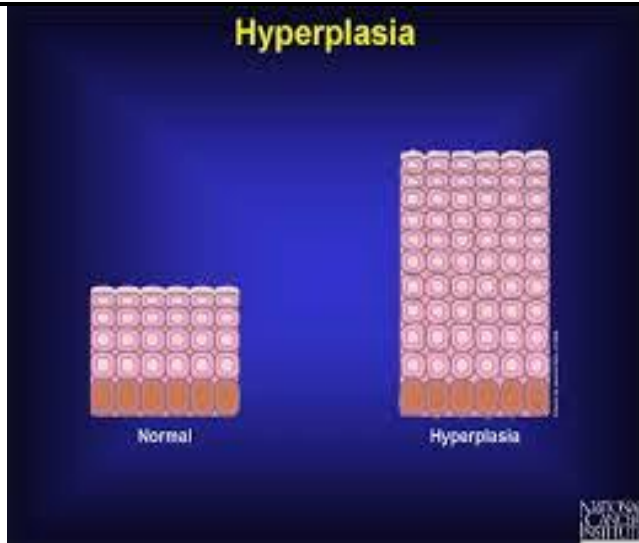
Intestinal wall hypertrophy:



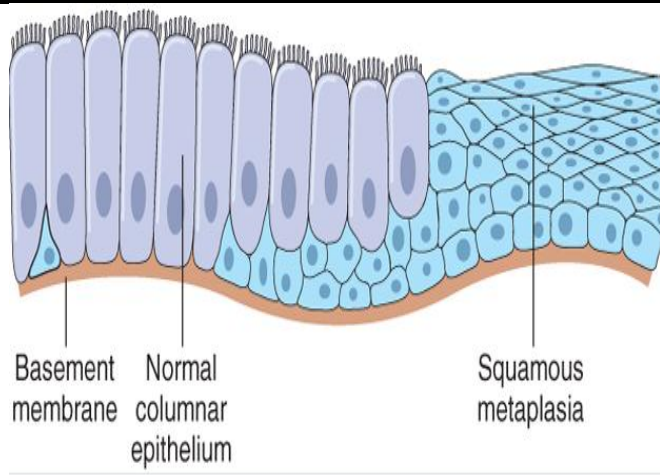
Stomach hypertrophy:



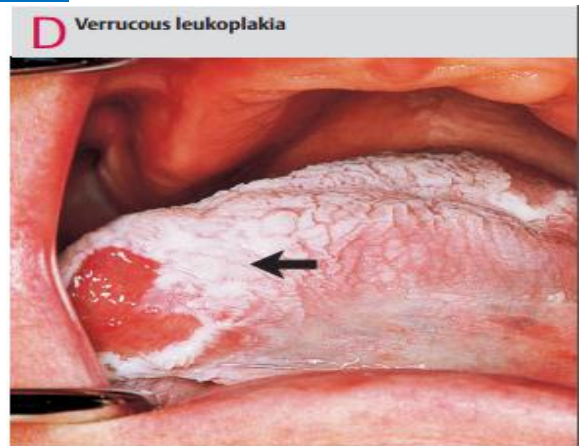
Hyperplasia:



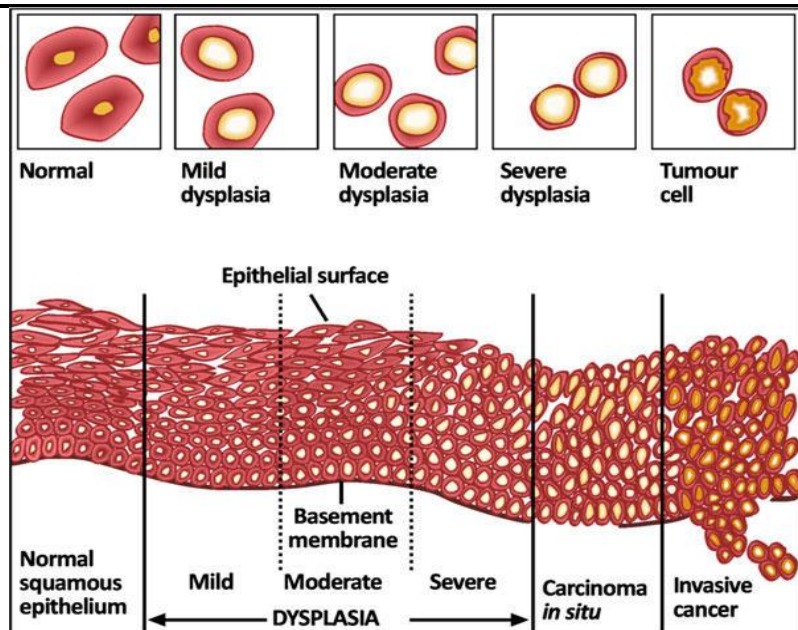
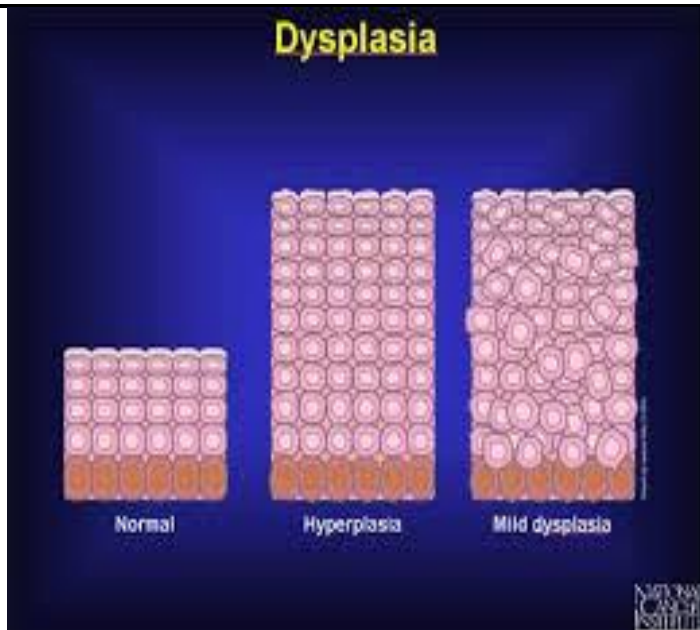
Metaplasia



Leukoplakia

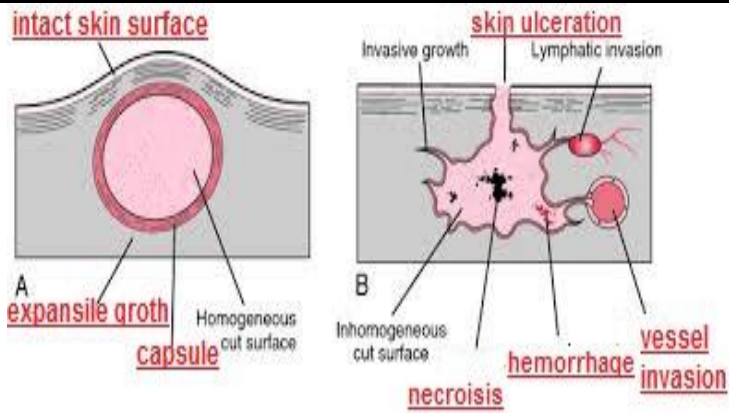


Dysplasia and CIN

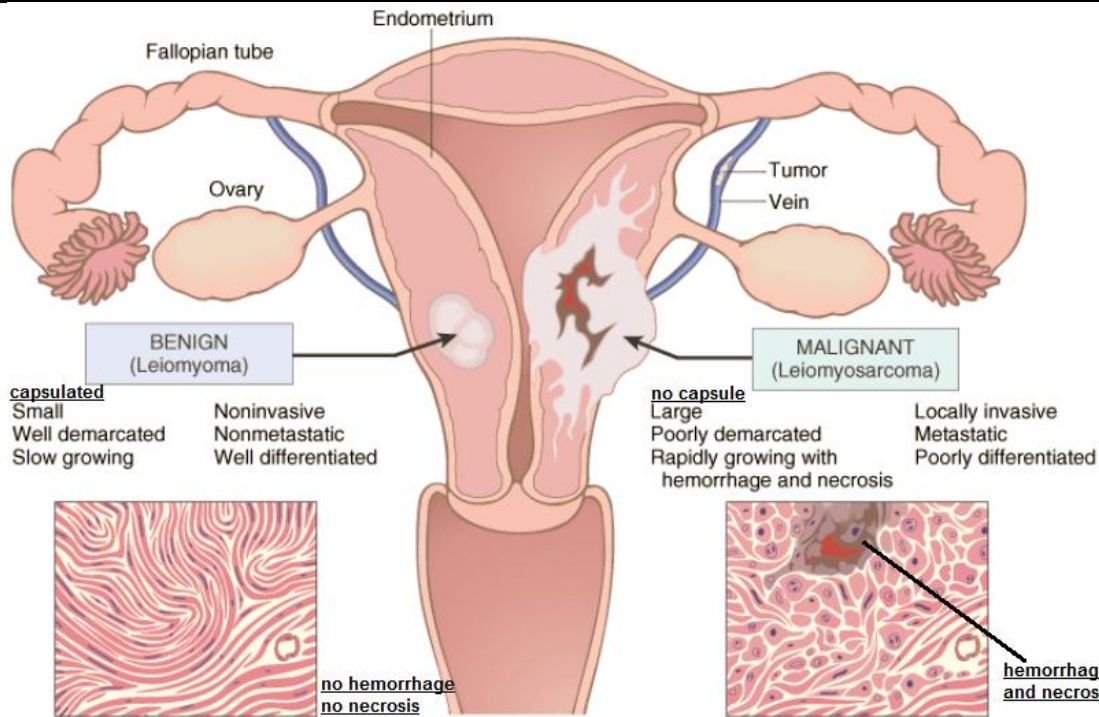
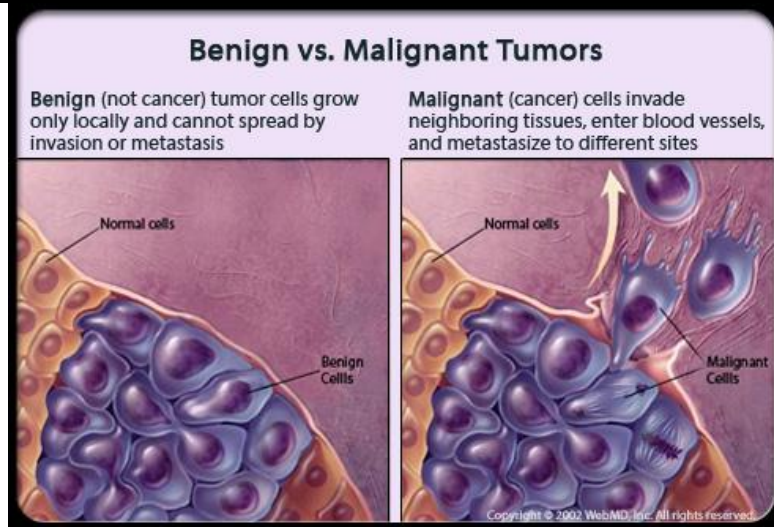


Neoplasia (tumors)

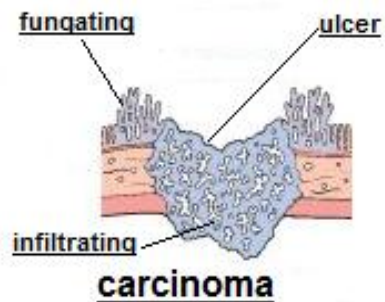
Characters of benign and malignant tumor

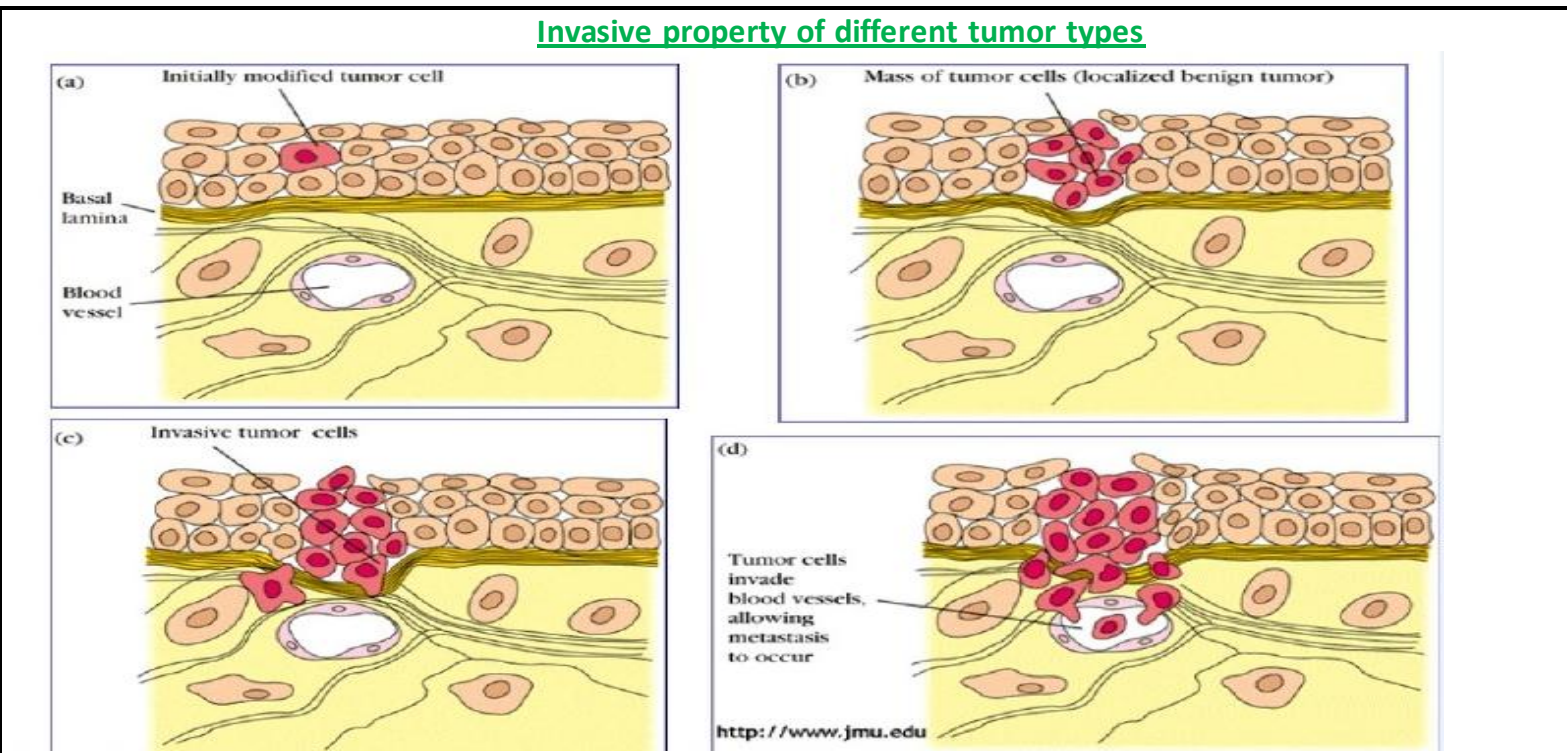
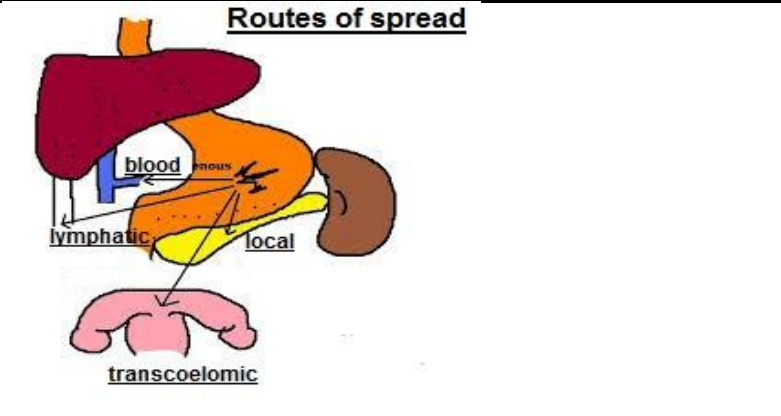
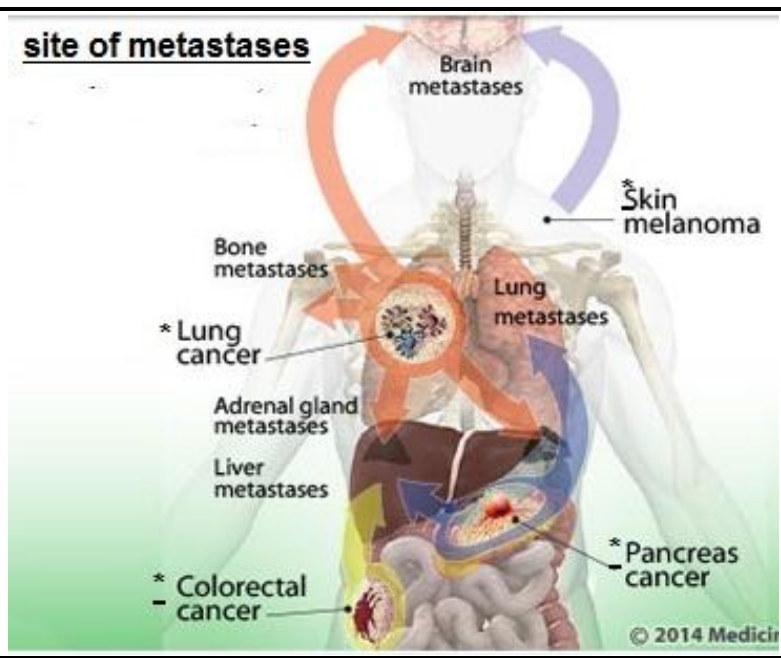
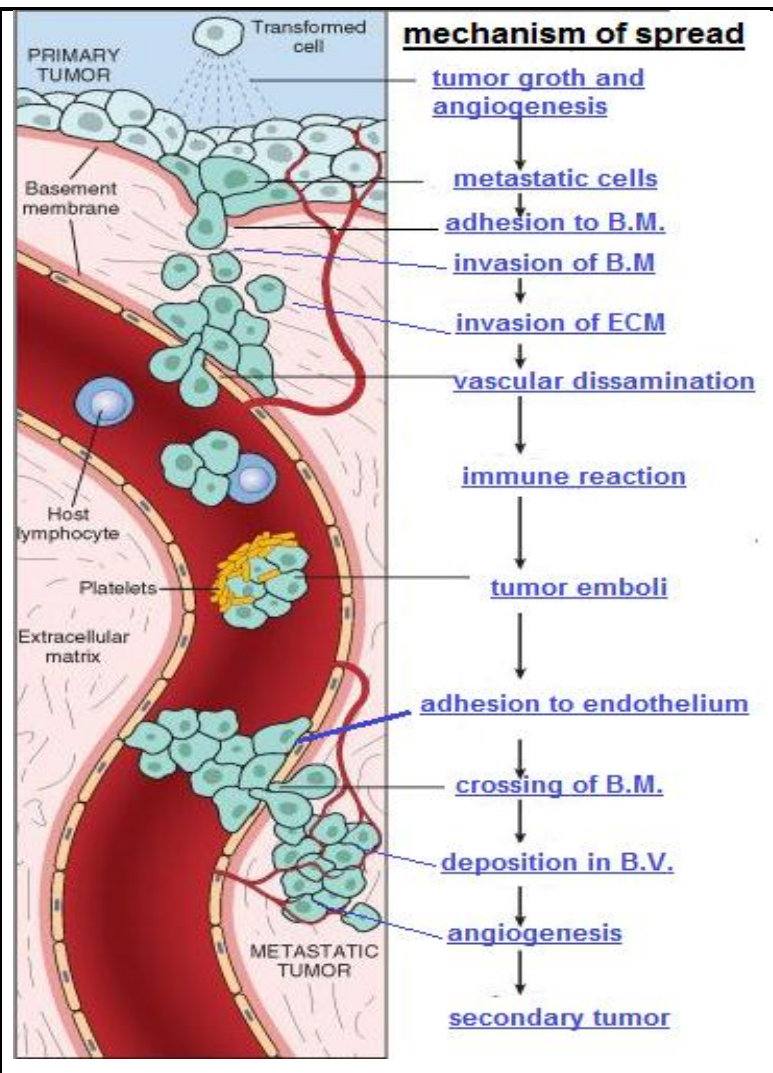


benign and malignant tumors

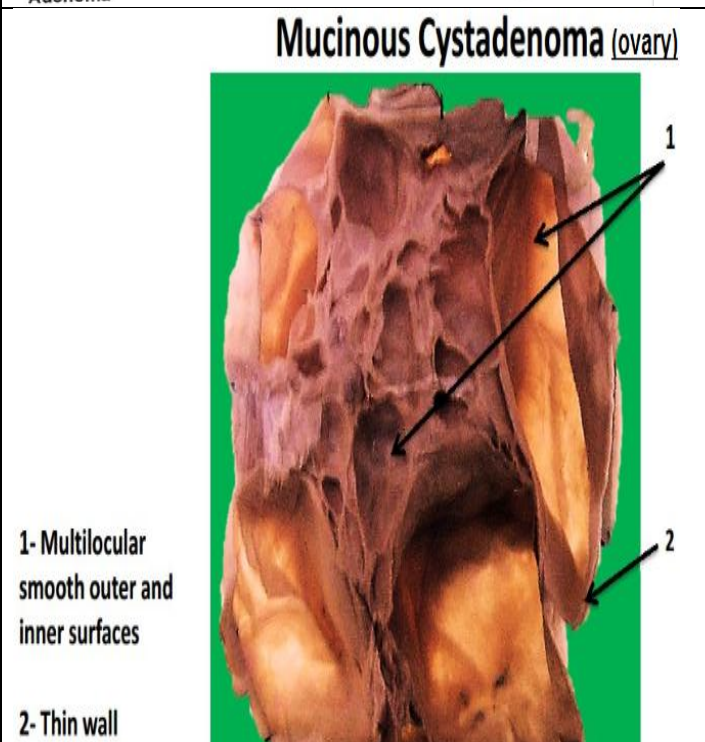
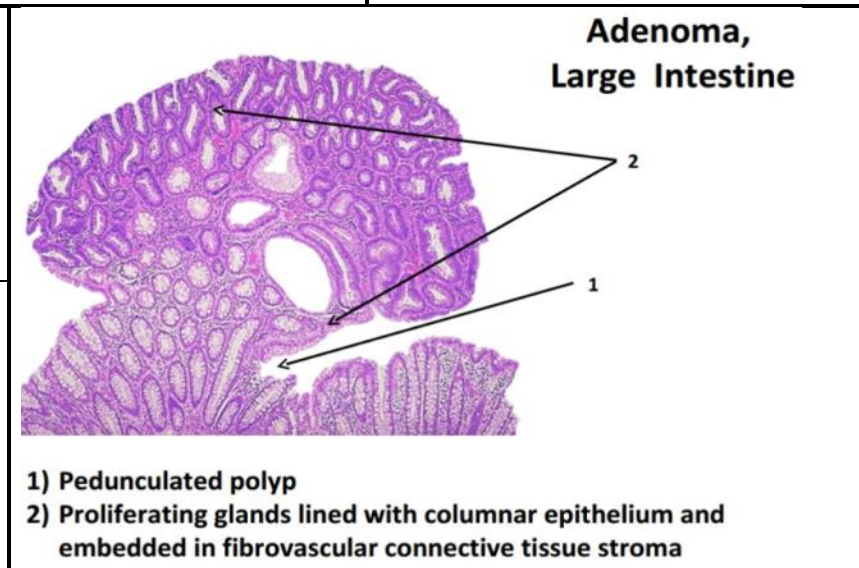
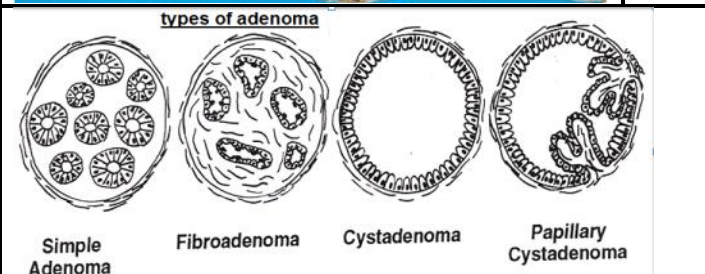
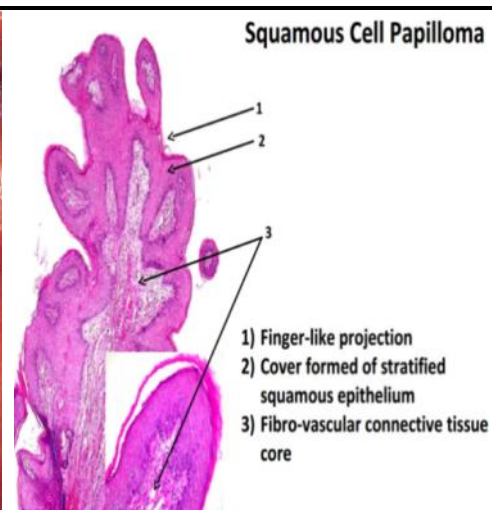
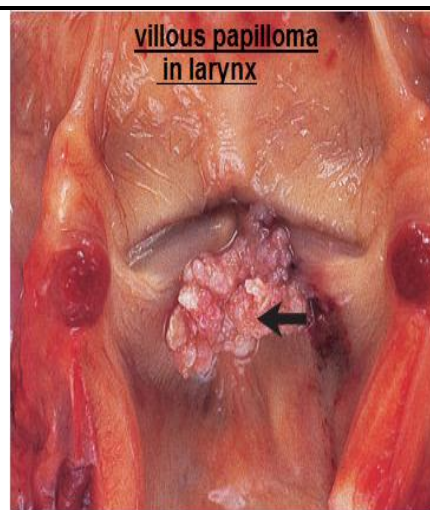
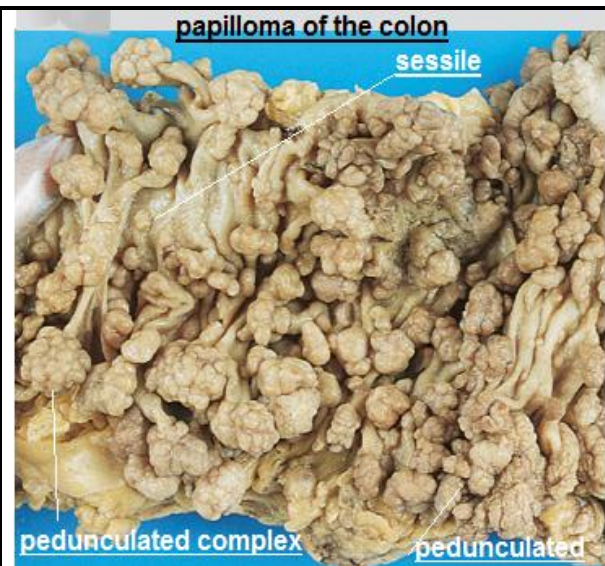


comparison between benign and malignant tumors



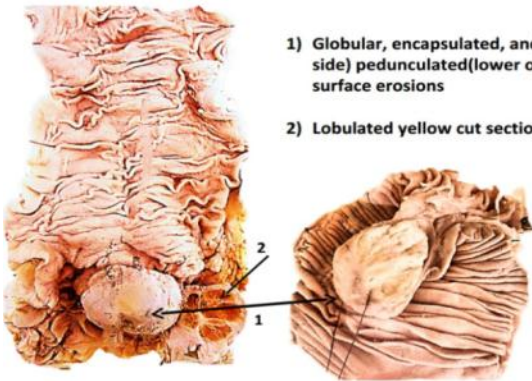


Papilloma and adenoma

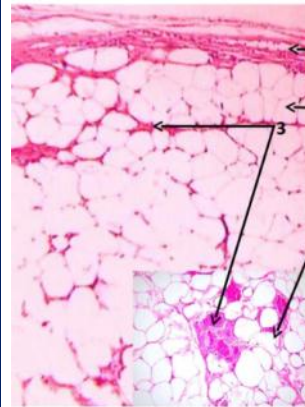


Lipoma (INTESTINE)

- 1) Globular, encapsulated, and sessile (left side) pedunculated (lower one) mass with surface erosions
- 2) Lobulated yellow cut section



Lipoma



- 1) Fibrous capsule
- 2) Lobules of mature, signet ring fat cells
- 3) Fibrovascular septa

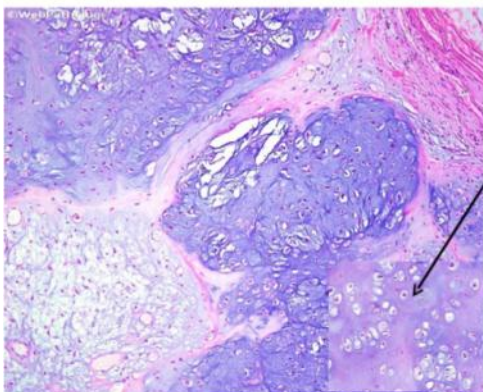


Fibroma



chondroma

Chondroma



- 1) Fibrohyaline capsule
- 2) Fibrovascular septa with areas of myxoid degeneration
- 3) Lobules of blue matrix containing chondrocytes arranged in groups of 2-4 cells with central dark nuclei and vacuolated-clear cytoplasm

fibromatosis in skin

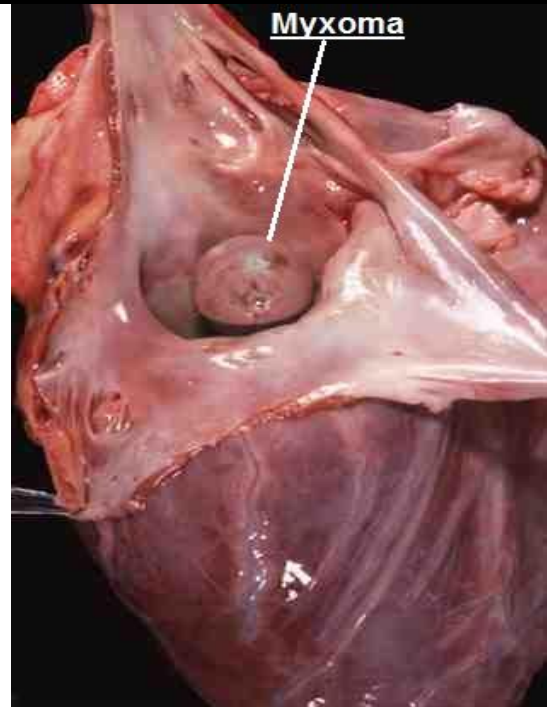


Exostosis (Osteochondroma)

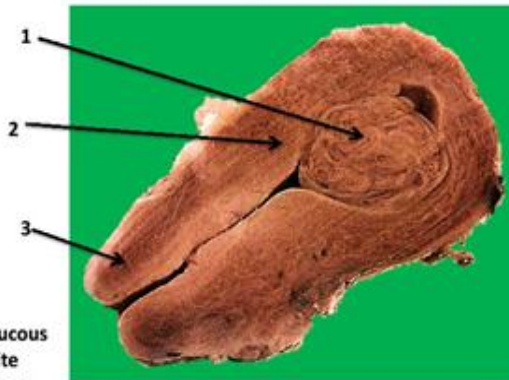


- 1) A- A projecting mass in the upper end of fibula at metaphysis in relation to epiphyseal cartilage.
B- The mass is formed of cancellous bone and covered by cartilage

Myxoma

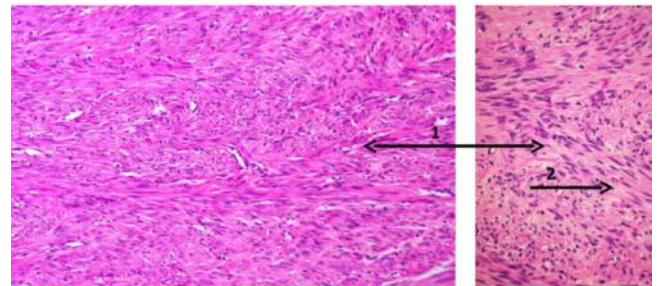


Submucous Leiomyoma



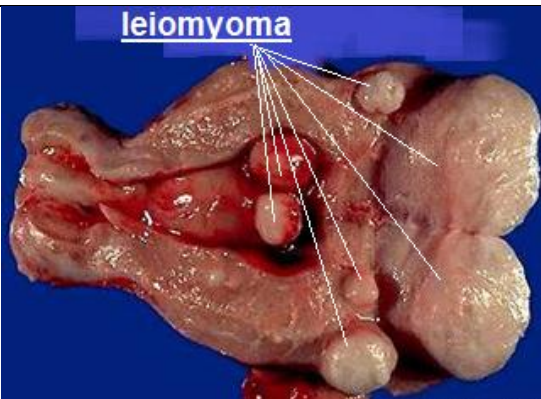
- 1- Fibroid; single submucous well-circumscribed white whorly uncapsulated mass
2- Myometrium
3- Cervix

Leiomyoma



- 1) Interlacing bundles of smooth muscle fibers and fibroblasts with areas of collagen
2) Smooth muscle fibers have elongated, cigar-shaped, round nuclei
No capsule

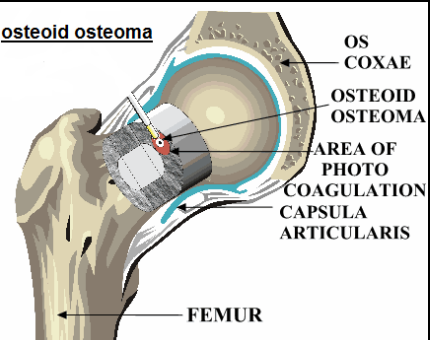
leiomyoma

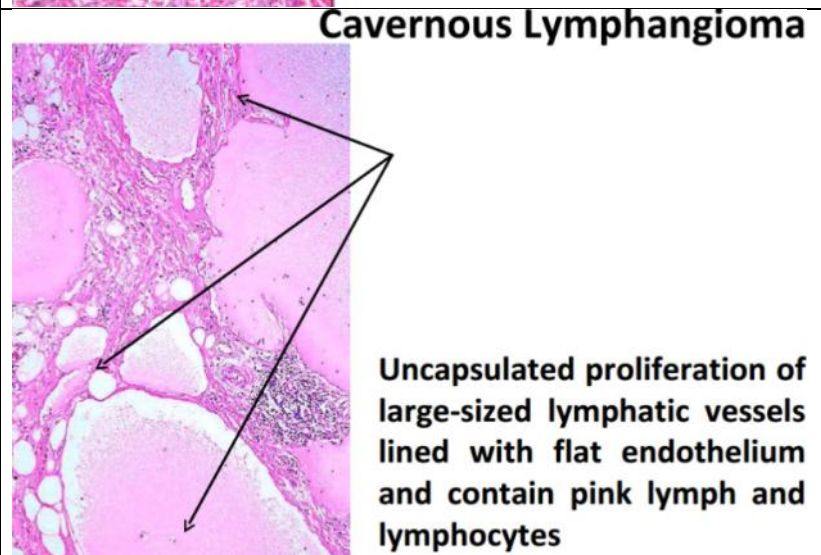
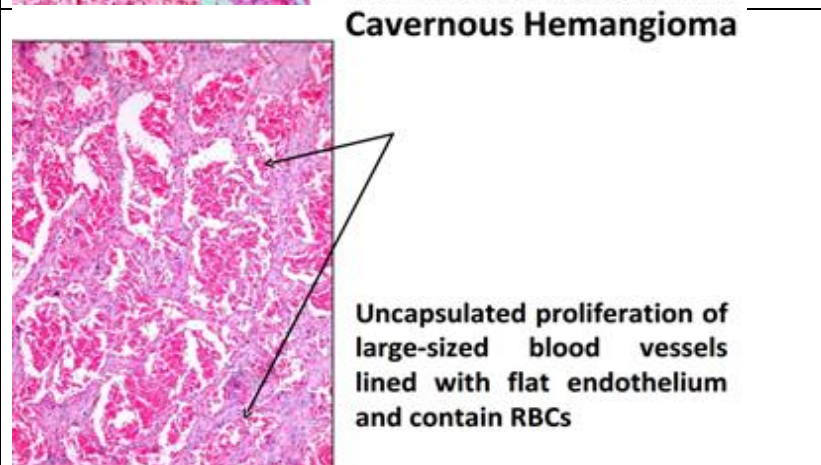
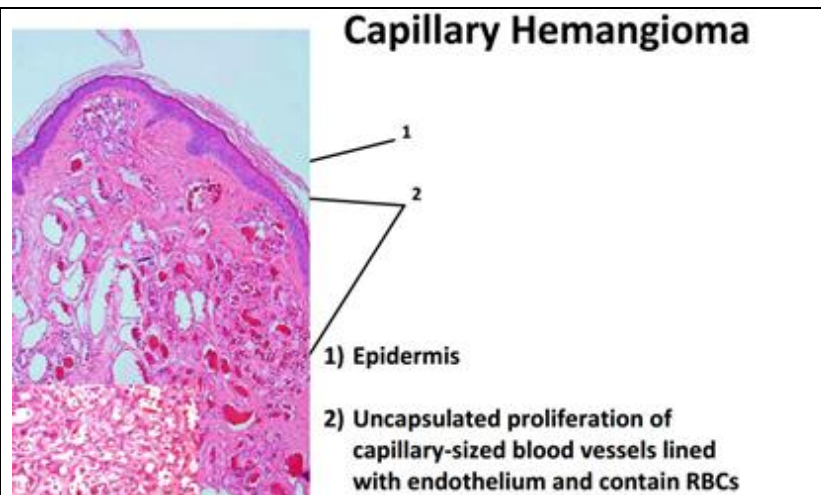


compact osteoma

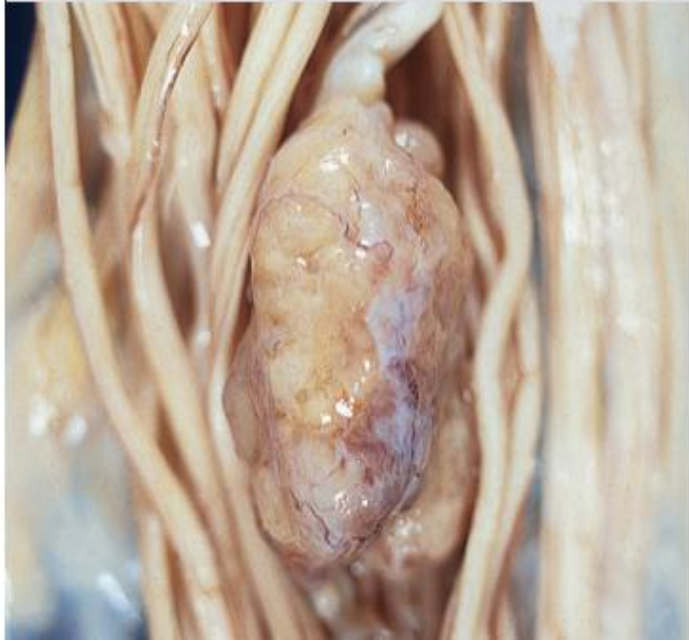


osteoid osteoma

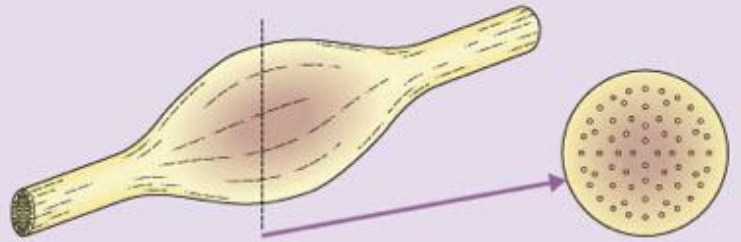




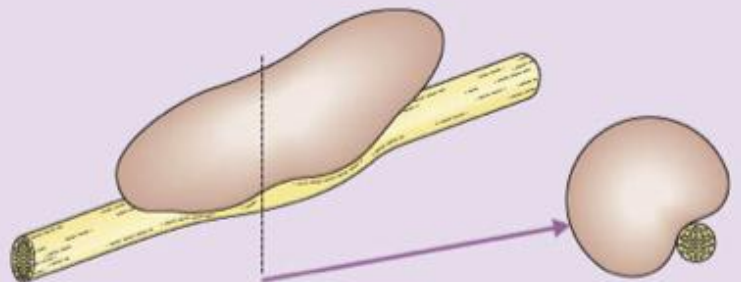
F Neurofibroma (spinal cord)



Neurofibroma



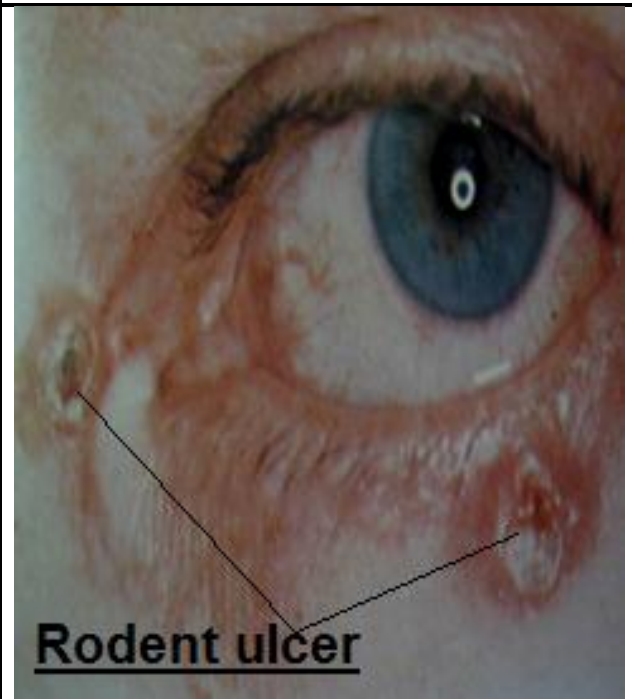
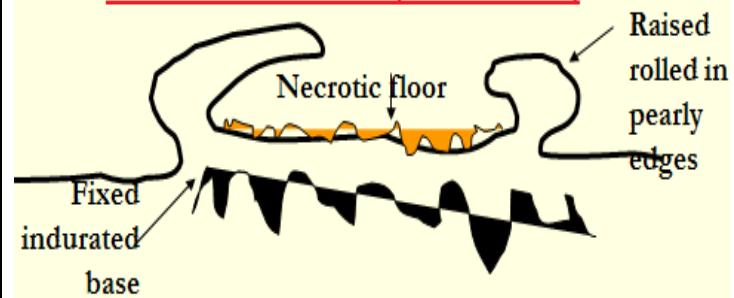
Schwannoma



squamous cell carcinoma



basal cell carcinoma (rodent ulcer)



Squamous cell carcinoma



Basal cell carcinoma



Squamous cell carcinoma

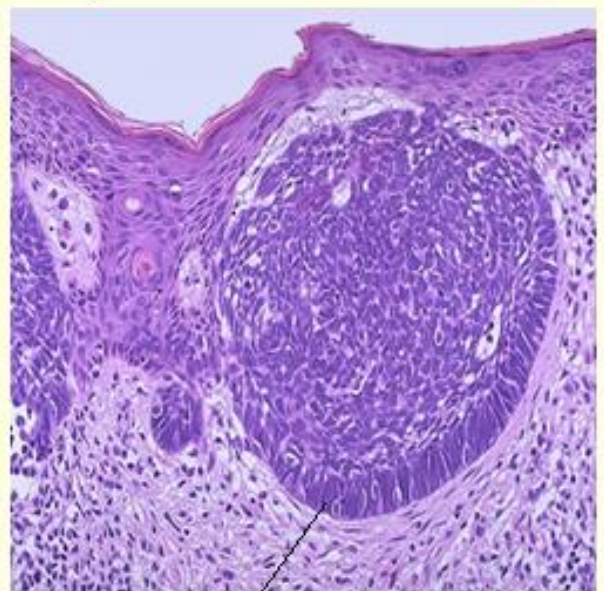
Squamous Cell Carcinoma



cell nests

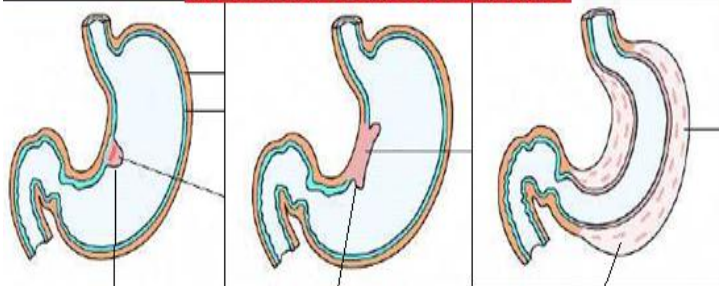
keratin

Basal Cell Carcinoma



palisading arrangement of cells

Carcinoma of mucosal surfaces

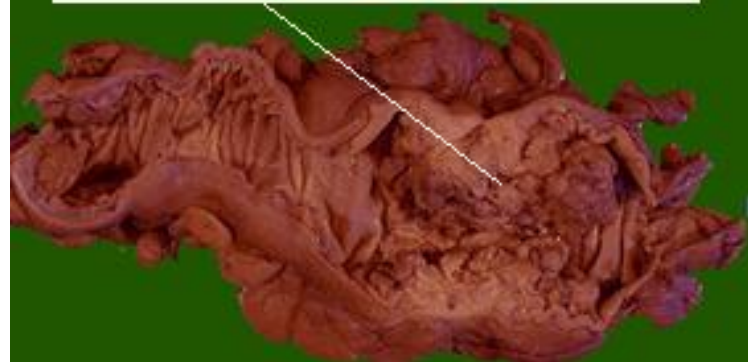


fungating

ulcerating

infiltrating

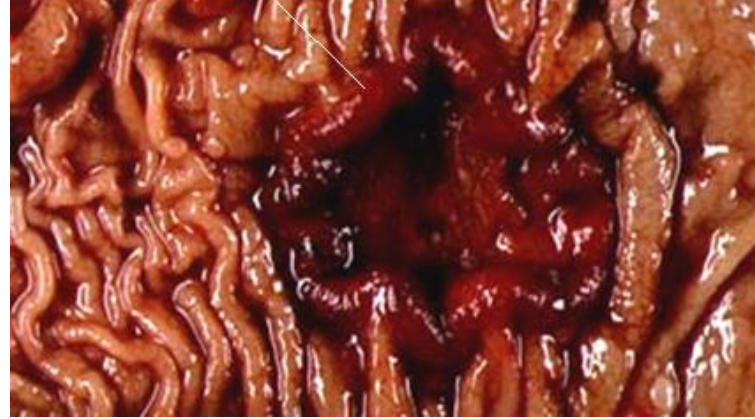
FUNGATING carcinoma of colon



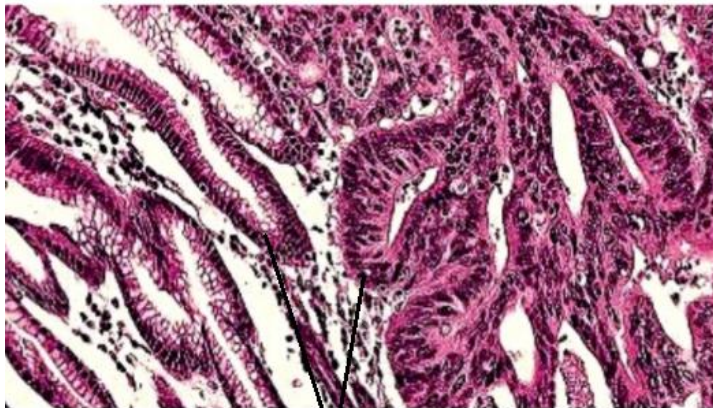
INFILTRATING carcinoma of stomach



ULCERATING carcinoma of stomach



Adenocarcinoma, Colon

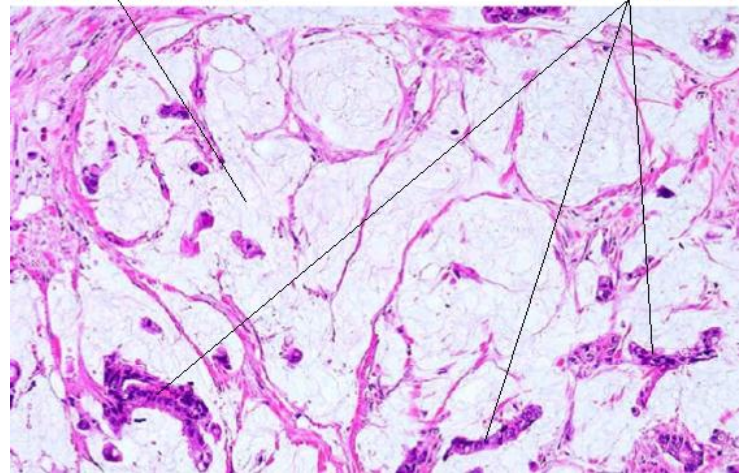


malignant gland lined by malignant cells

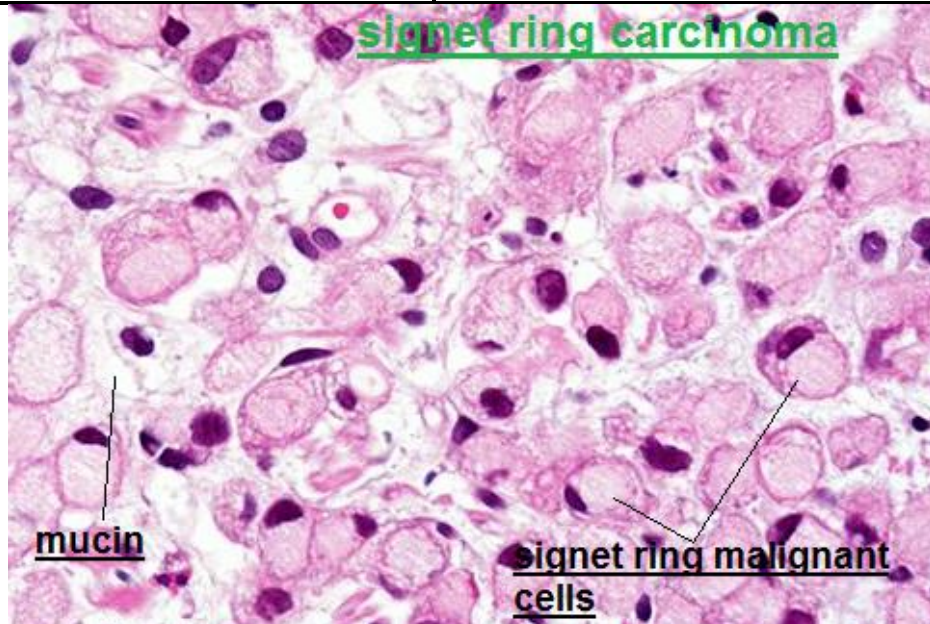
Mucoid Carcinoma

pale mucinous pools

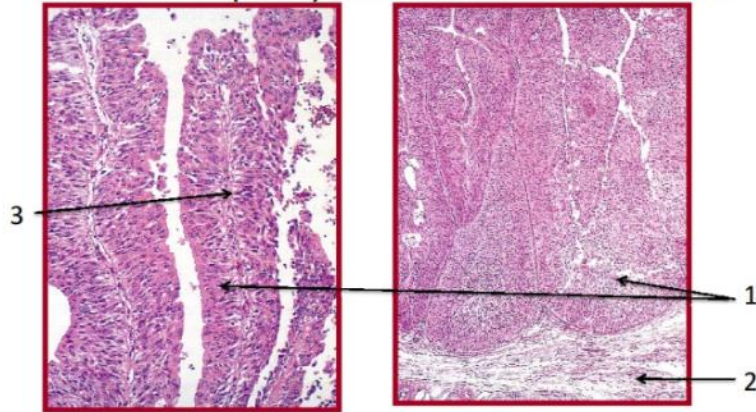
malignant glands



signet ring carcinoma



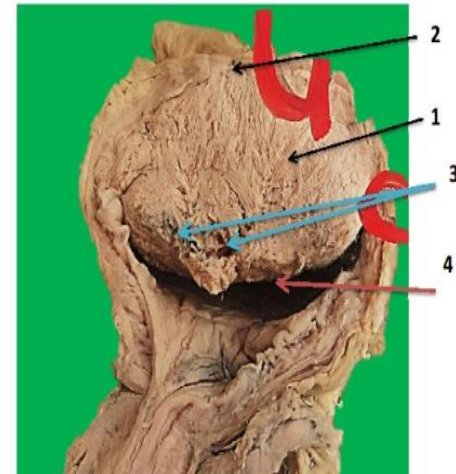
Papillary transitional cell carcinoma



- 1- Malignant cells forming papillae lined with multiple more than 8 malignant transitional cells with enlarged hyperchromatic pleomorphic nuclei.
- 2- Lamina propria.
- 3- Connective tissue core

Polypoid carcinoma of urinary bladder

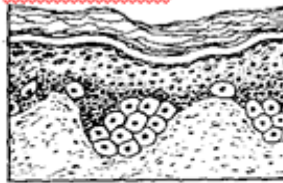
- 1- malignant polypoid tumor mass filling the bladder cavity
- 2- the tumor is infiltrating the bladder wall
- 3- Areas of hemorrhage and necrosis
- 4- Surface ulceration



pigmented nevus

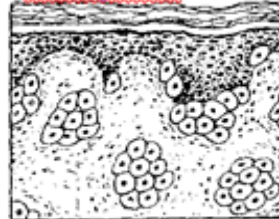


Junctional nevus



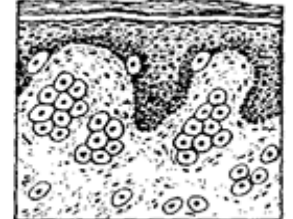
Proliferation at dermoepidermal junction

Compound nevus



Proliferation in dermis and junctional areas

Intradermal nevus

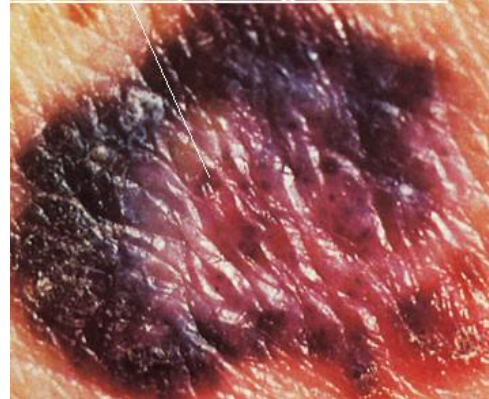


Proliferation is dermis only

lentigo melanoma



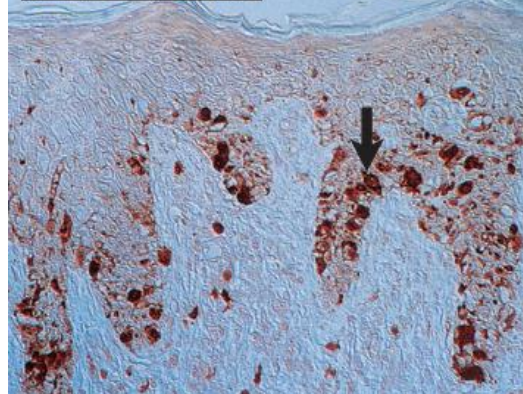
superficial spreading melanoma



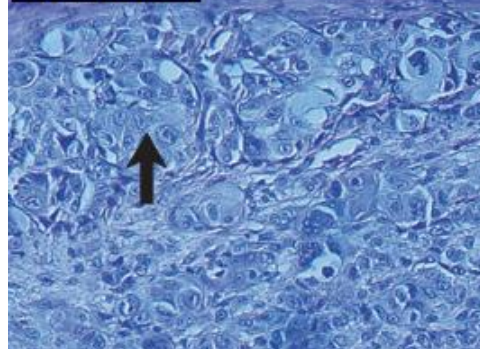
nodular melanoma



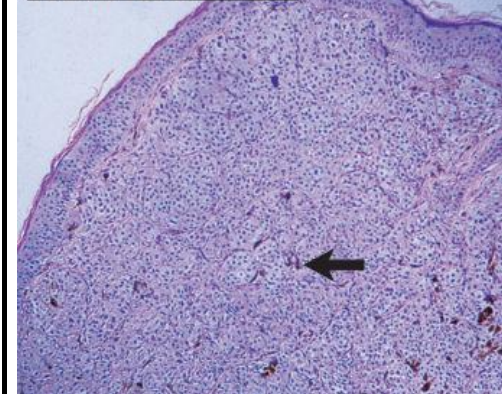
lentigo melanoma



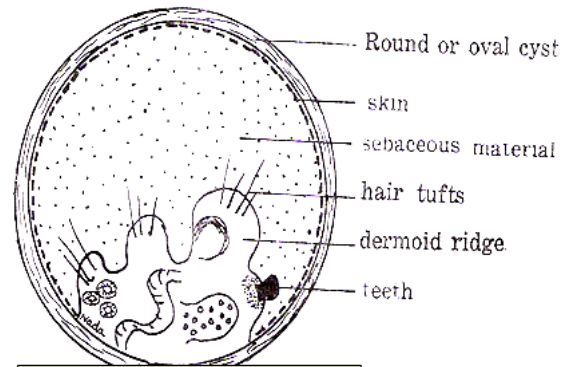
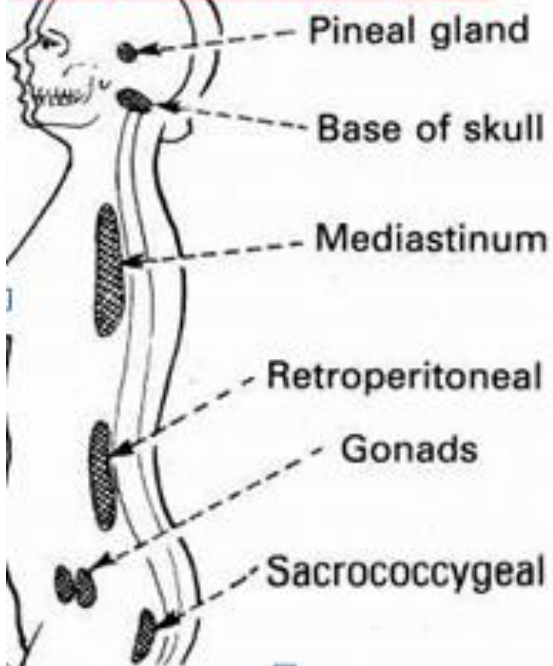
superficial spreading melanoma



nodular melanoma



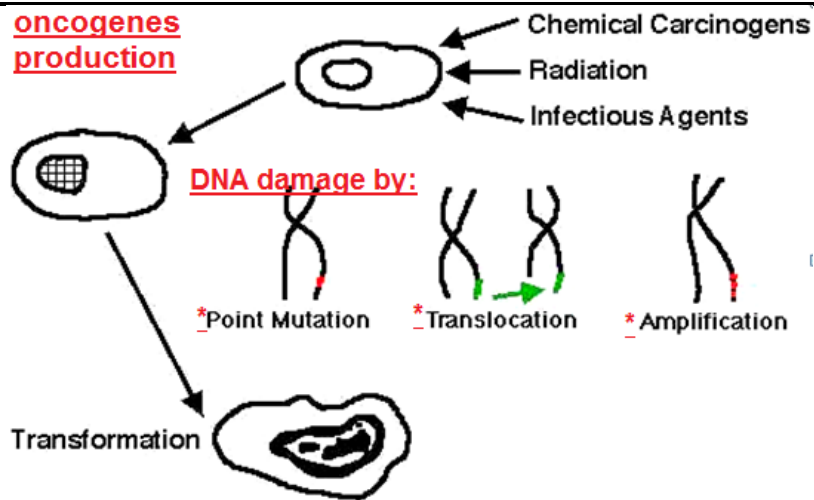
sites of teratoma



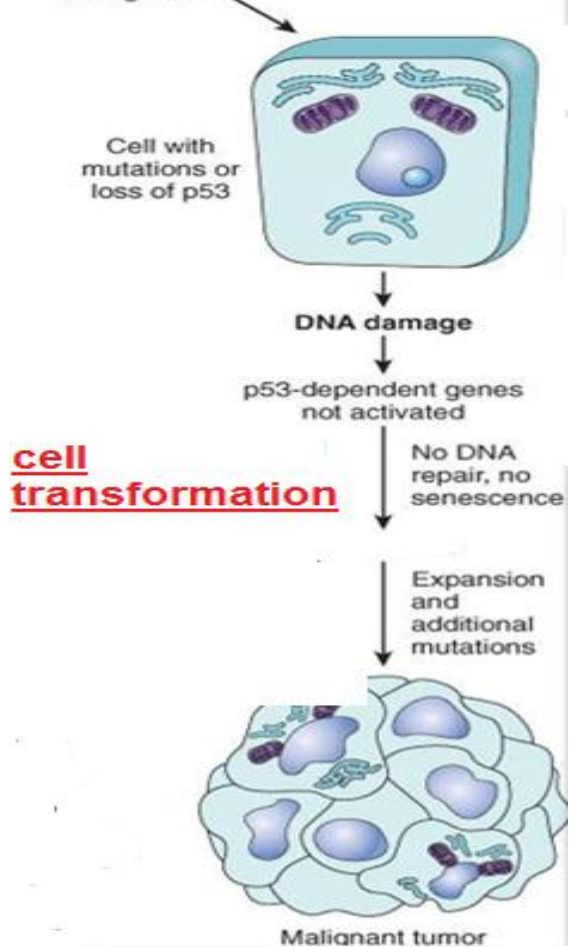
Dermoid Cyst



oncogenes production

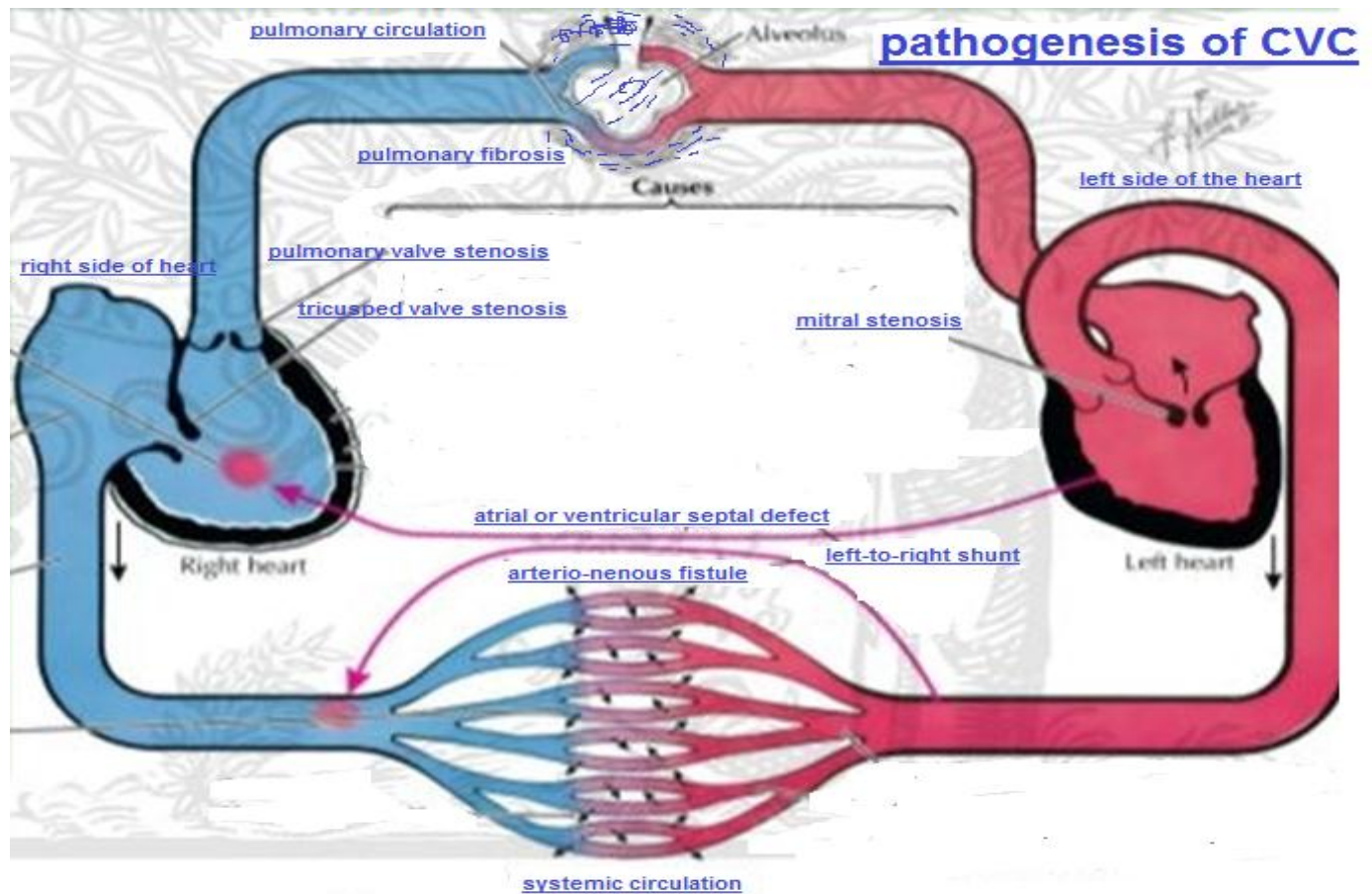


Ionizing radiation
Carcinogens
Mutagens



cell transformation

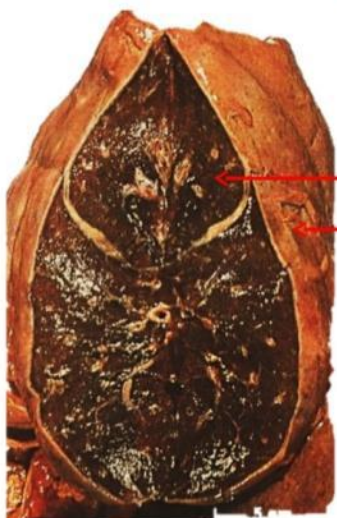
Blood disorders



CVC lung

Gross picture

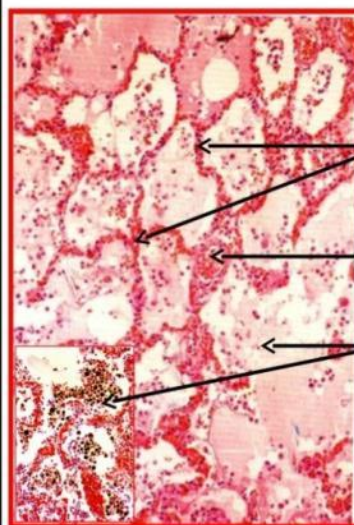
Chronic Venous Congestion (CVC) (Brown Induration) of Lung



- 1
- 2
- 1) Firm, brown, and consolidated lung tissue
- 2) Thick fibrous pleura

Microscopic picture

Chronic Venous Congestion, Lung

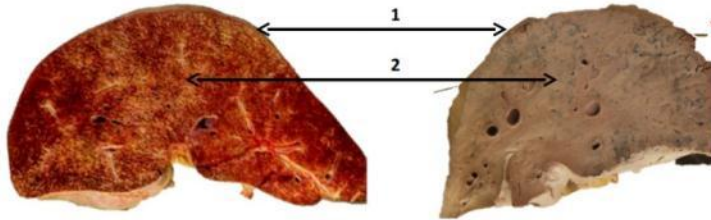


- 1
- 2
- 3
- 1) Thickened, edematous alveolar walls containing RBCs
- 2) Alveolar spaces containing edema fluid, disintegrated RBCs, and "heart failure cells" (inset)
- 3) Thickened, edematous alveolar septum containing congested blood vessels

CVC liver

Gross picture

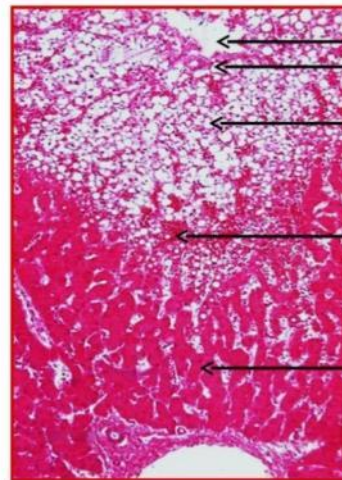
Chronic Venous Congestion, Liver (Nut-meg Liver)



- 1) Sharp border (indicating firmness of tissue)
- 2) Variegated, mottled cut section representing congested areas (dark) alternating with areas of fatty change (yellow-pink)

Microscopic picture

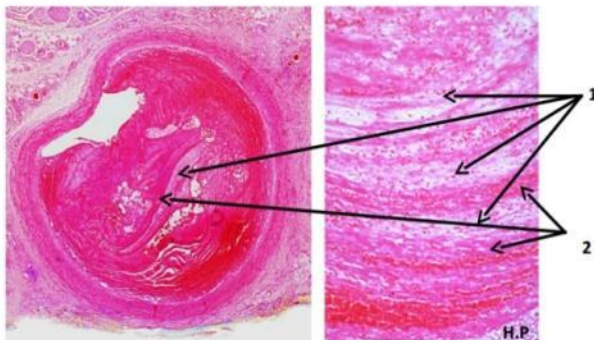
Chronic Venous Congestion, Liver



- 1) Dilated central vein
- 2) Necrotic hepatocytes
- 3) Fatty change of hepatocytes
- 4) Atrophic hepatocytes with fat vacuoles
- 5) Normal-looking hepatocytes

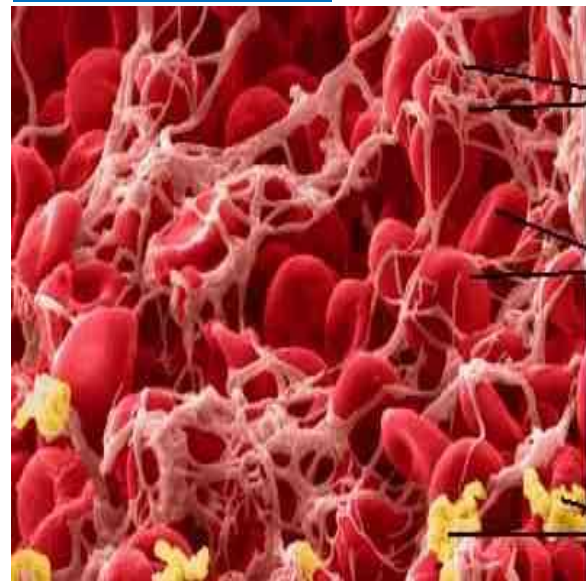
Recent thrombus

Recent Thrombus



- 1) Lines of Zahn (fused platelets)
- 2) Fibrin threads admixed with white and red cells

Structure of thrombus

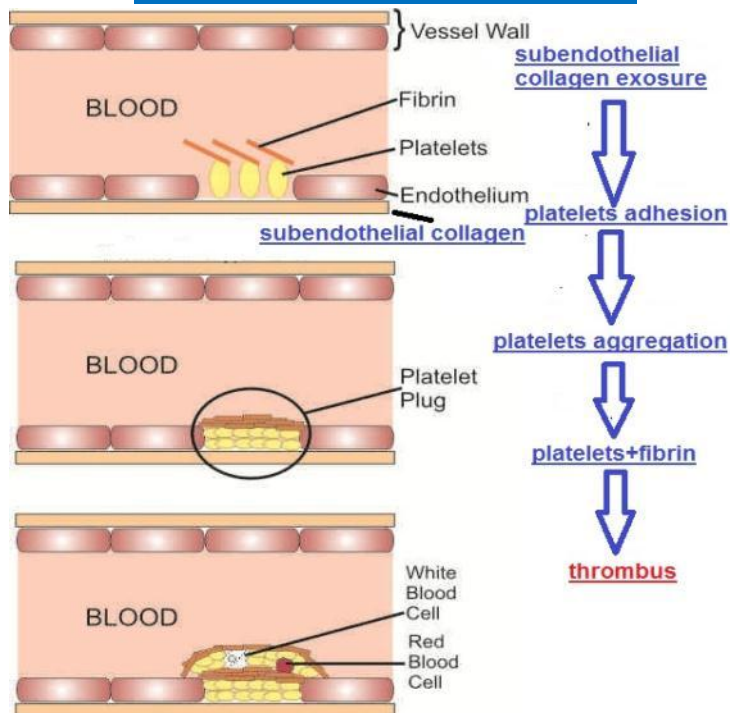


Fibrin

Red cells

Platelets

Mechanism of thrombus formation



Thrombus size

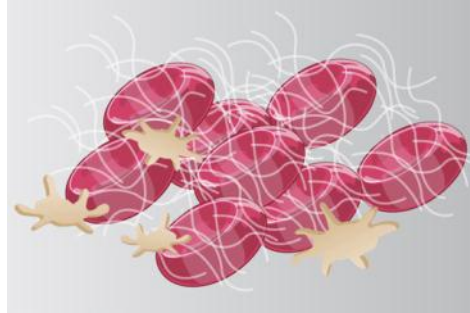


PROPAGATING THROMBUS



Thrombus color

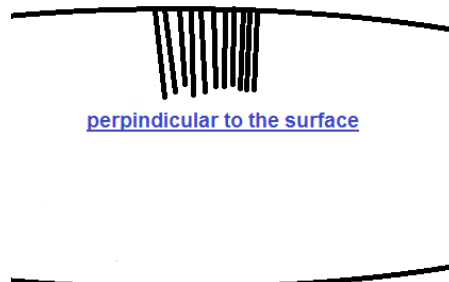
Red thrombi (more RBCs- little platelets)



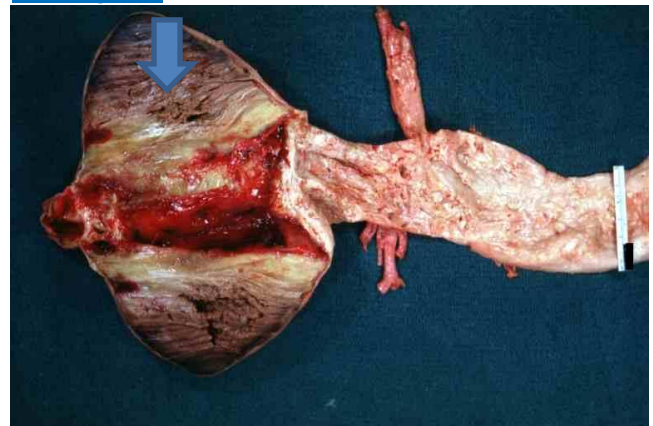
Pale thrombi (more platelets- little RBC)



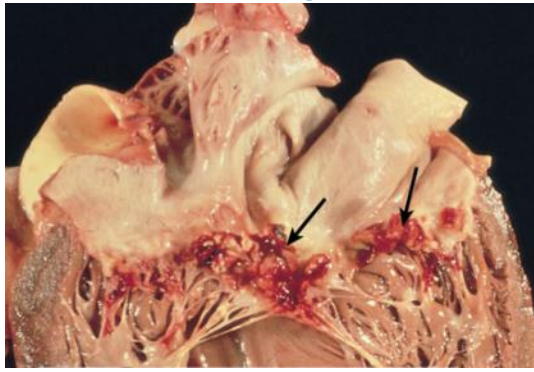
Mixed coralline thrombus



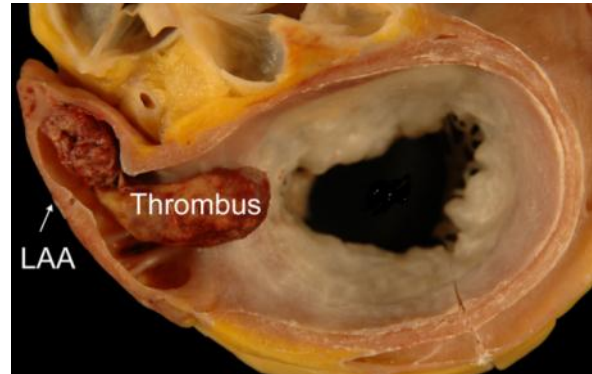
Mixed laminated thrombus (opened aortic aneurysm)



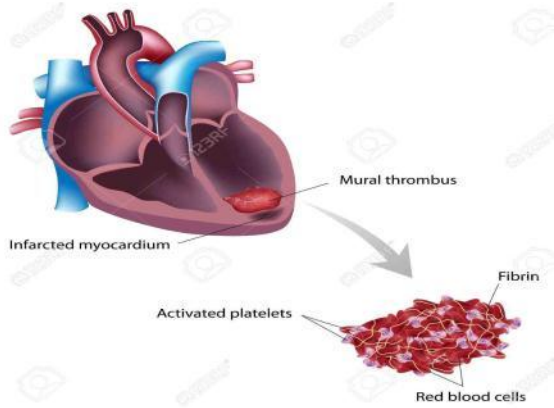
Valvular thrombi (vegetation)



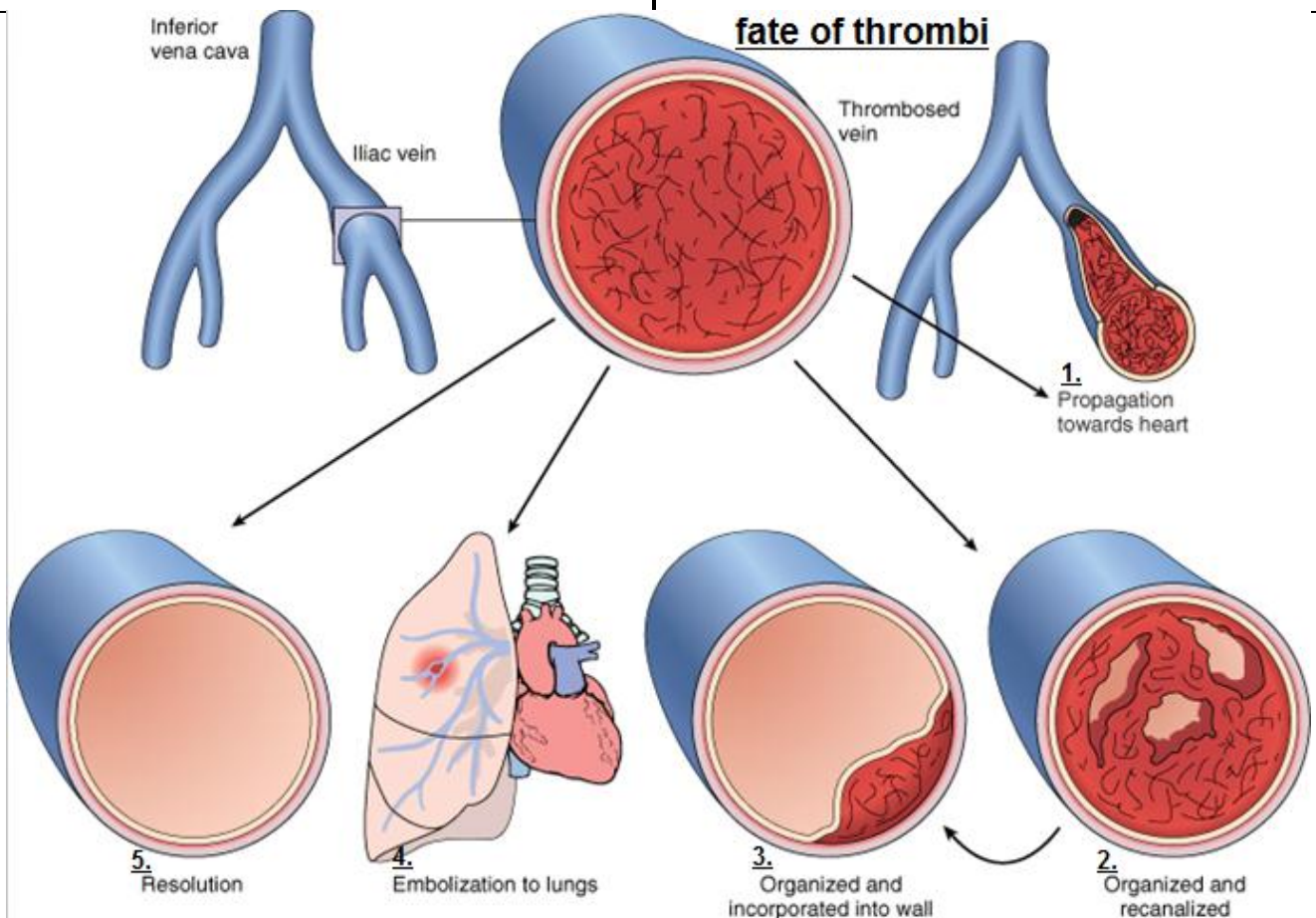
Atrial thrombi



Ventricular mural thrombus



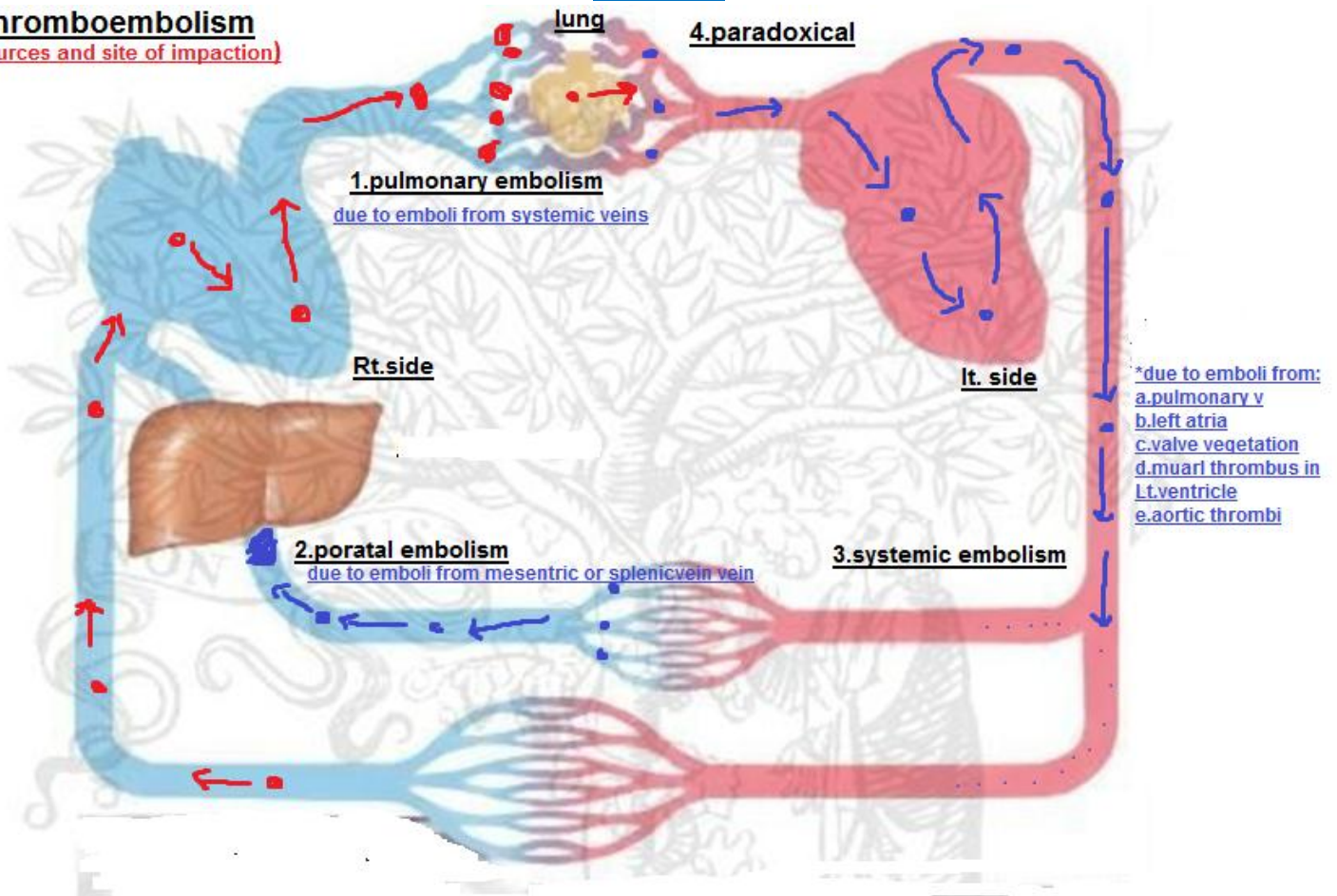
Venous thrombi



Embolism

thromboembolism

(sources and site of impaction)

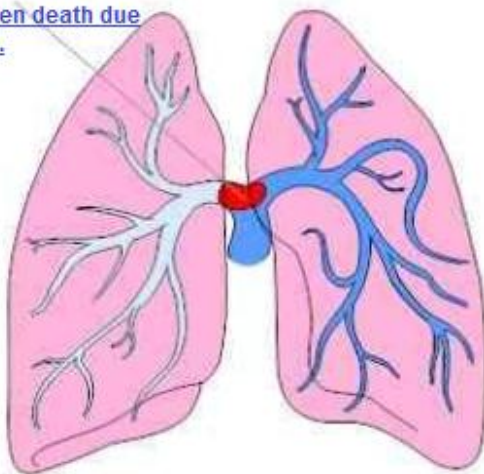


Pulmonary thromboembolism

site of impaction and effect of pulmonary embolism

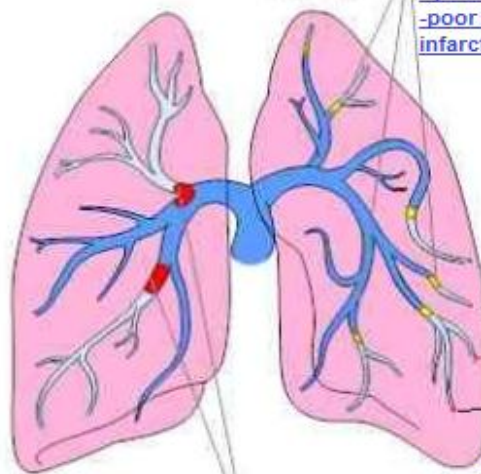
Pulmonary embolism in main pulmonary artery

cause sudden death due to acute H.F.



Multiple emboli in small branches of pulmonary artery

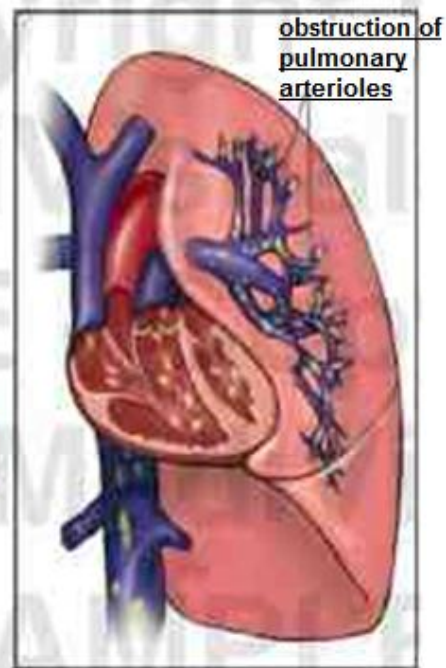
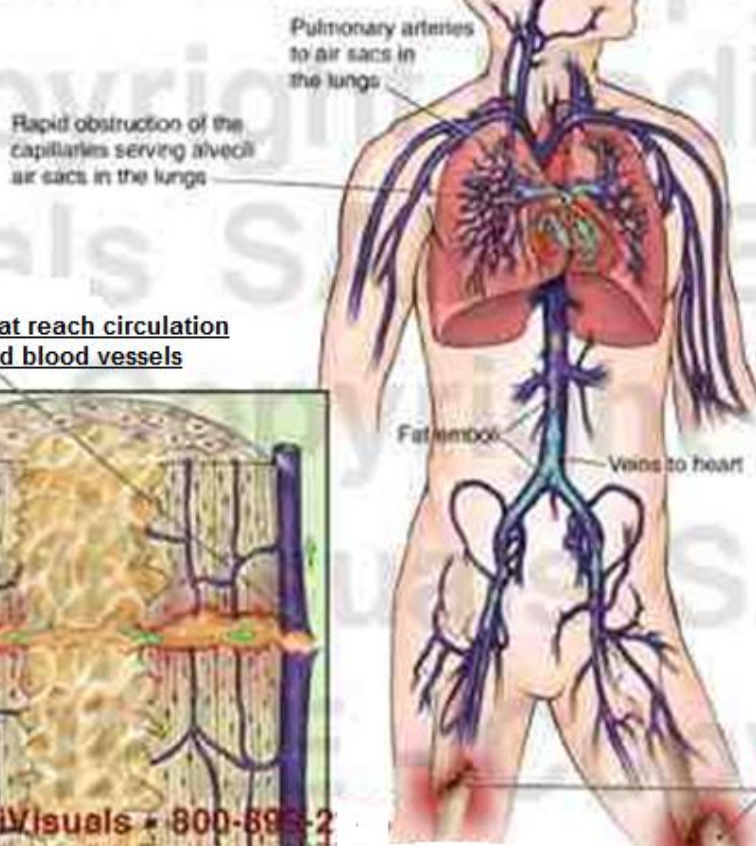
-good collaterals>>no effect
-poor collaterals>>infarction+gangrene



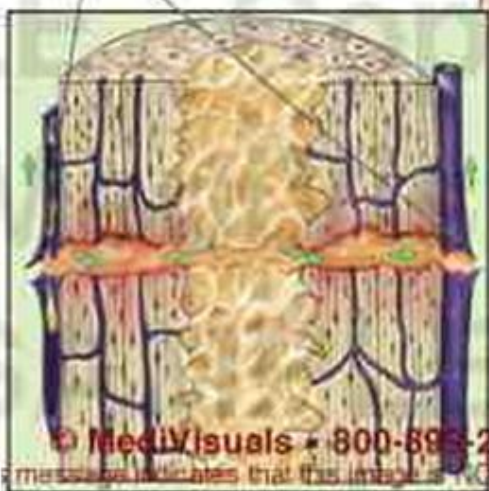
very small emboli
-no effect
-paradoxical embolism

Pulmonary emboli in main branches of pulmonary arteries

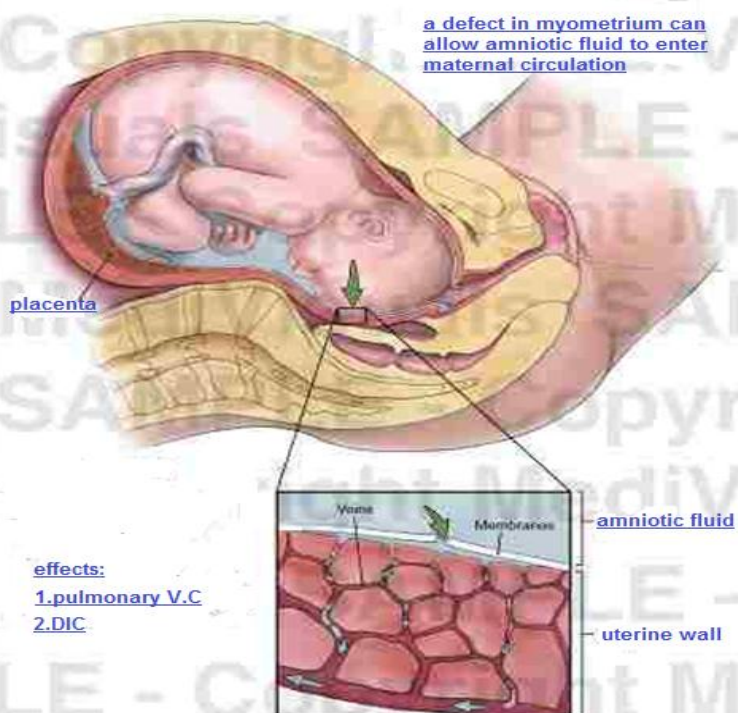
Fat Embolism



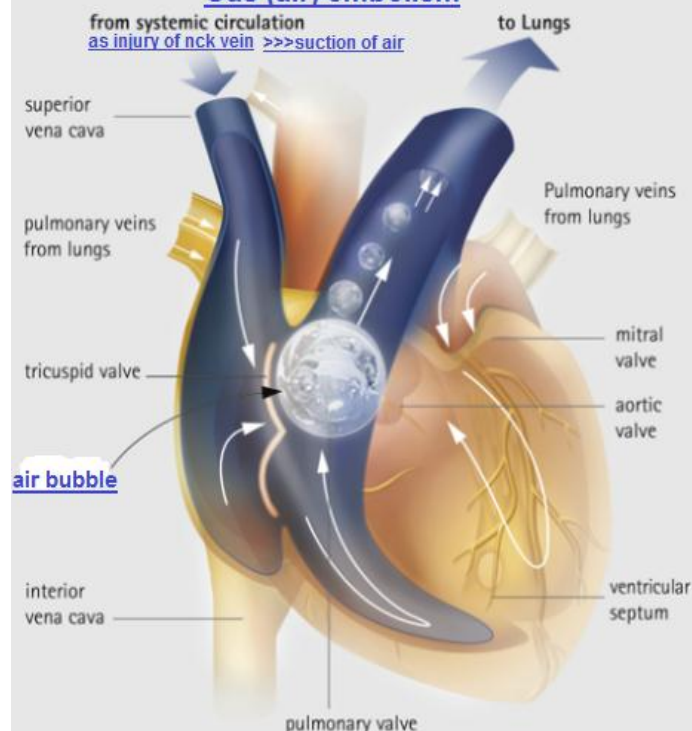
bone marrow fat reach circulation through injured blood vessels



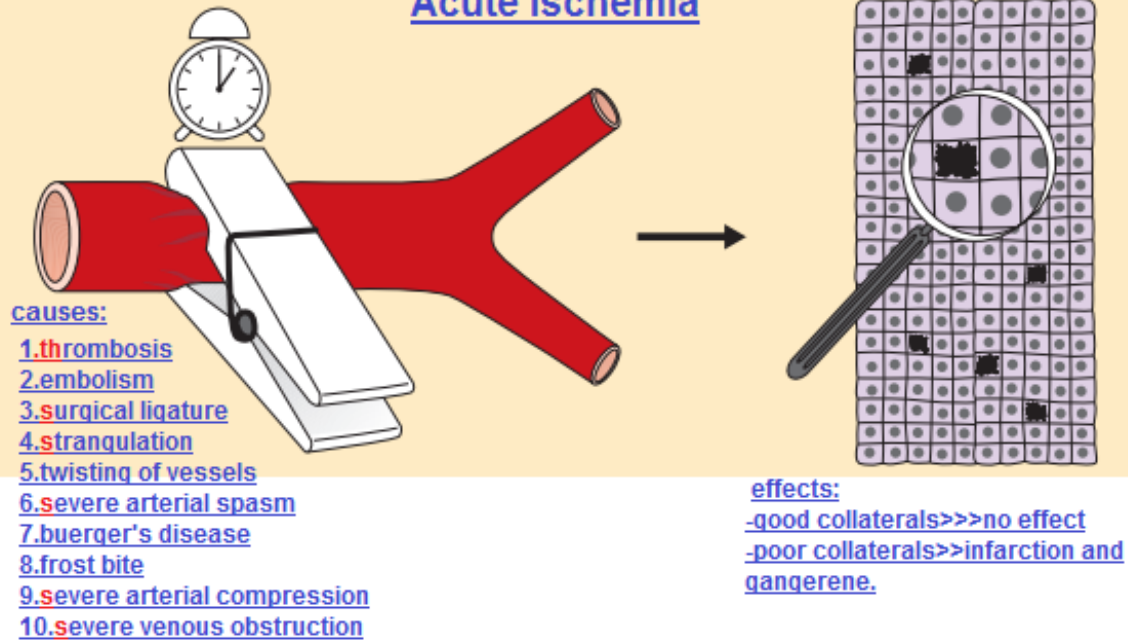
Amniotic Fluid Embolism



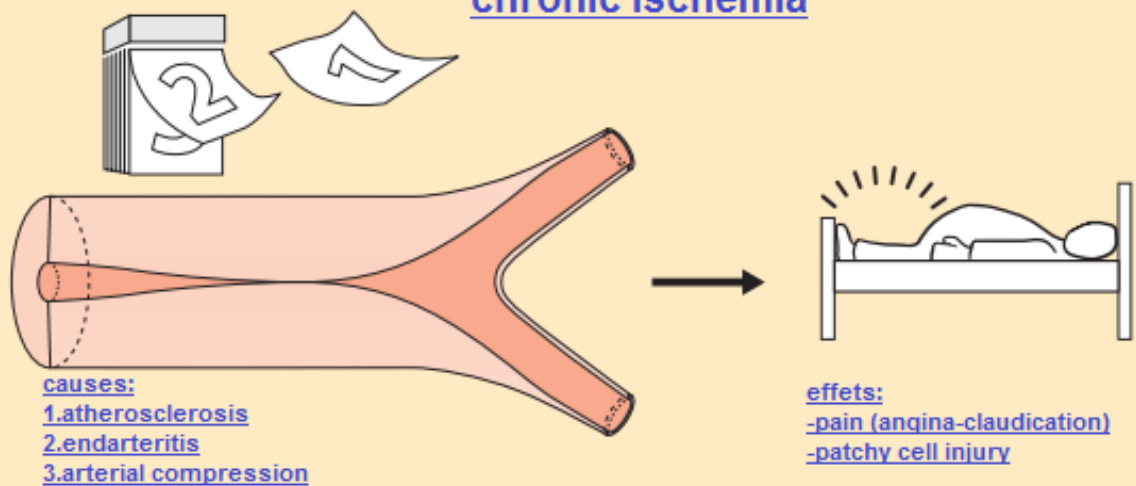
Gas (air) embolism



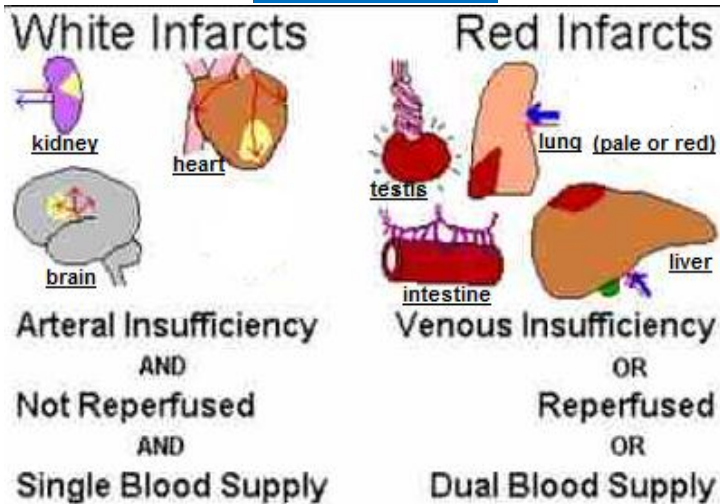
Acute ischemia



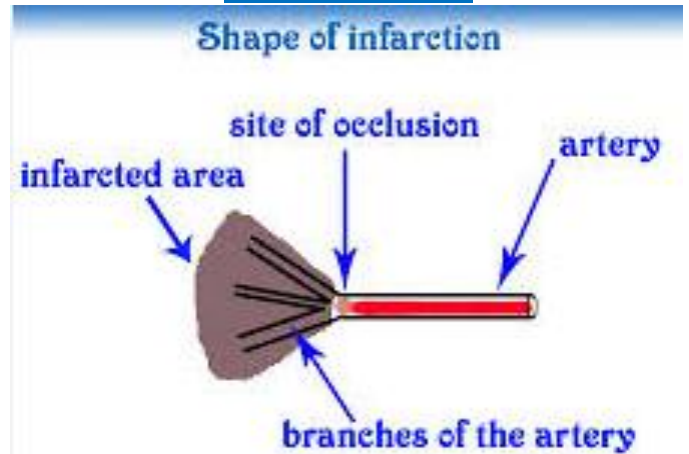
chronic ischemia



Types of infarct

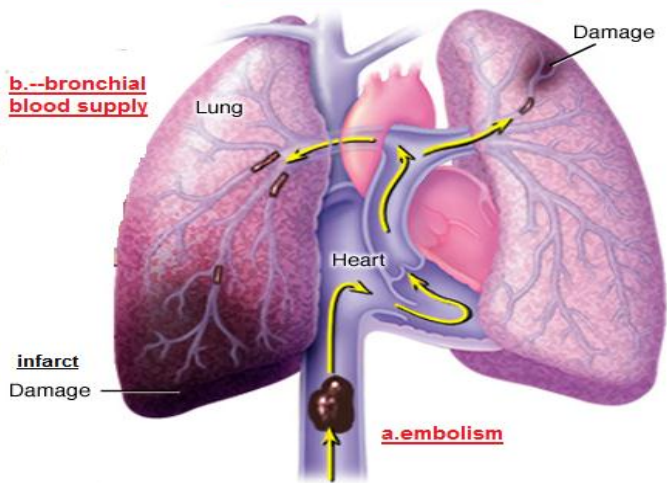


Shape of infarct



Lung infarction

aetiology of lung infarction

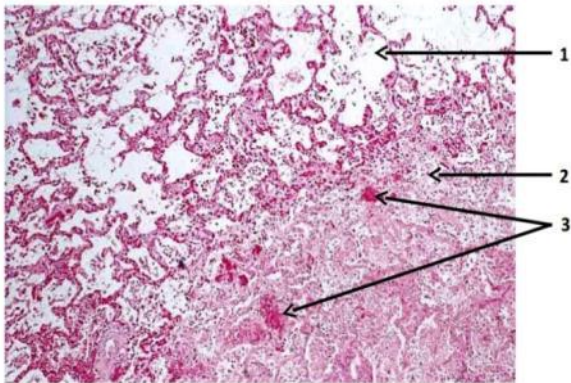


Gross picture



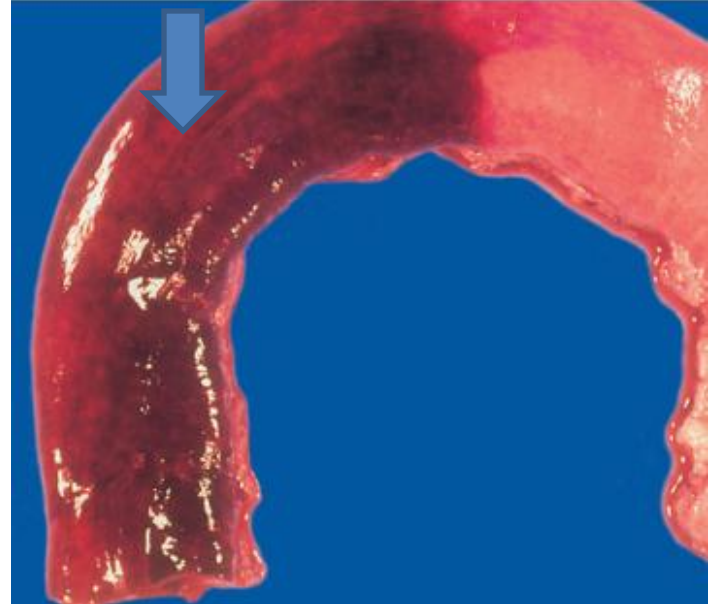
Microscopic picture

Hemorrhagic Lung Infarction



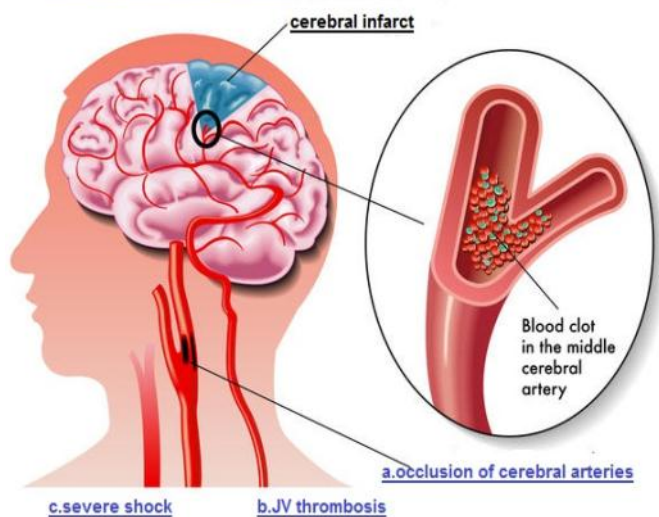
- 1) Area of CVC
- 2) Area of hemorrhagic infarction: ghosts of lung structures admixed with intact and hemolysed RBCs
- 3) Emboli in pulmonary artery branches

Intestinal infarction

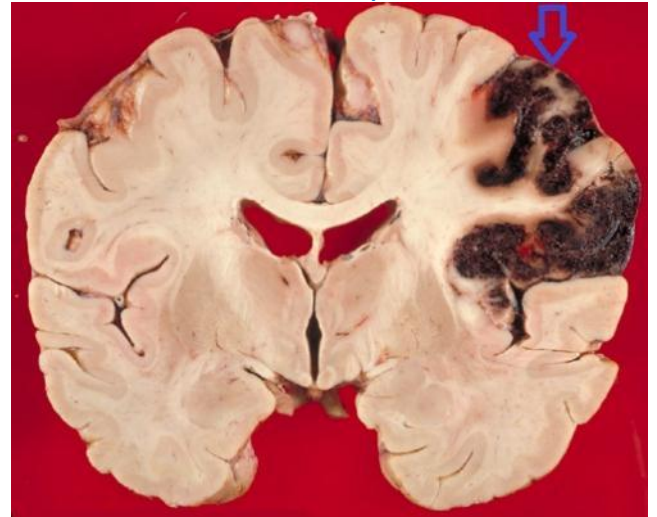


Cerebral infarction

aetiology of cerebral infarction

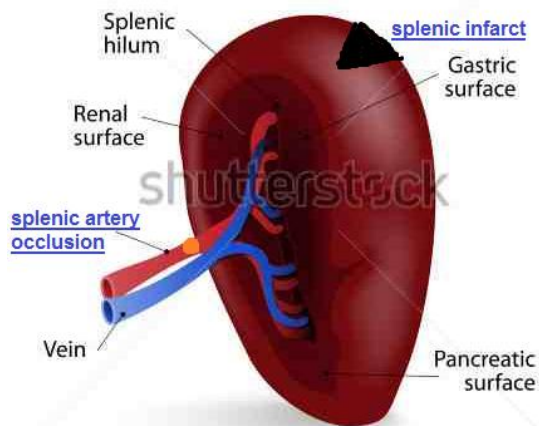


Gross picture

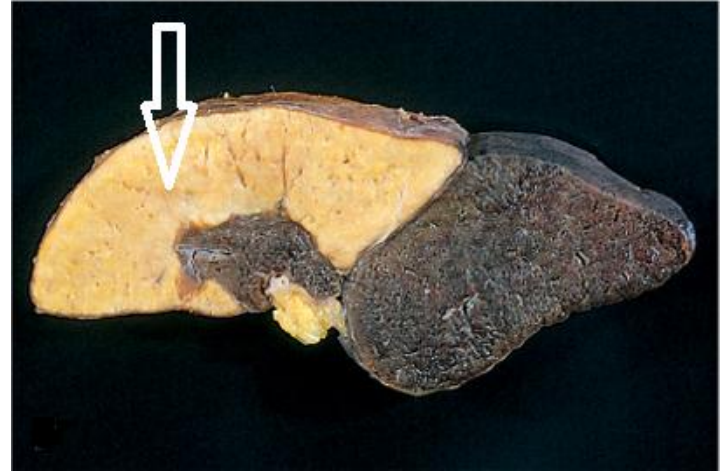


Splenic infarction

aetiology of splenic infarction

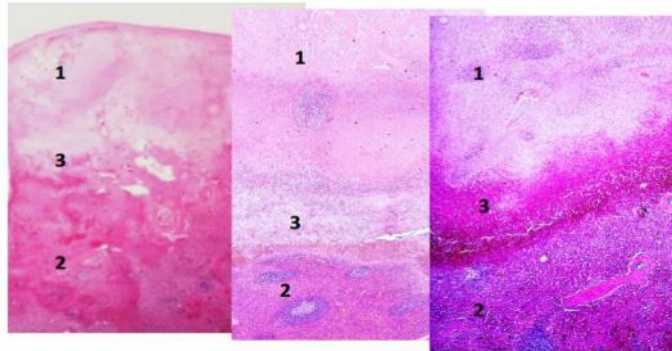


Gross picture



Microscopic picture

Infarction, Spleen



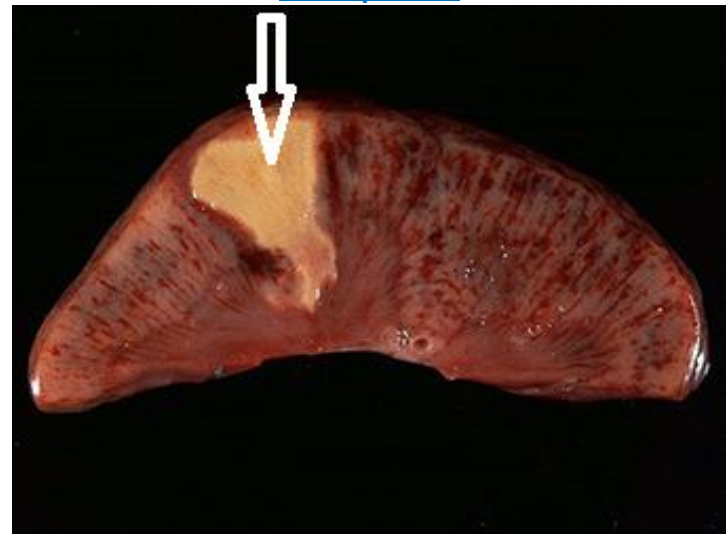
- 1) Zone of infarction
- 2) Normal spleen
- 3) Zone of congestion and granulation tissue

Kidney infarction

aetiology of renal infarction

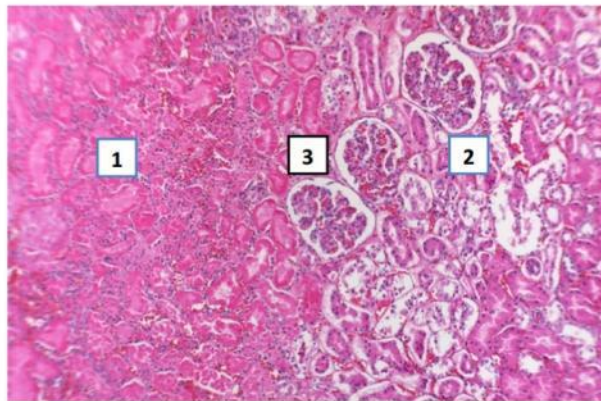


Gross picture



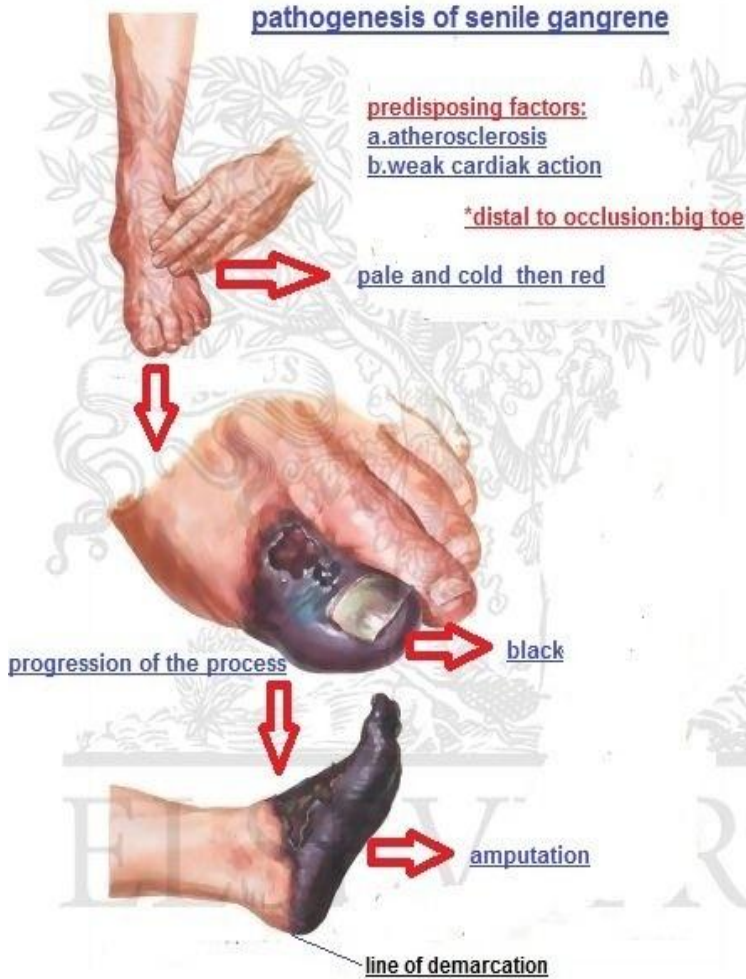
Microscopic picture

Infarction Kidney



- 1) Zone of infarction
- 2) Normal kidney
- 3) Zone of congestion and granulation tissue

pathogenesis of senile gangrene



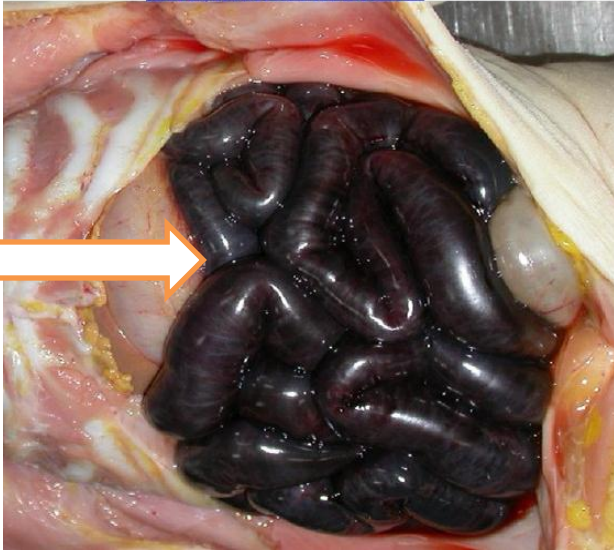
dry gangrene



wet gangrene



intestinal gangrene



diabetic gangrene



Cancrum oris

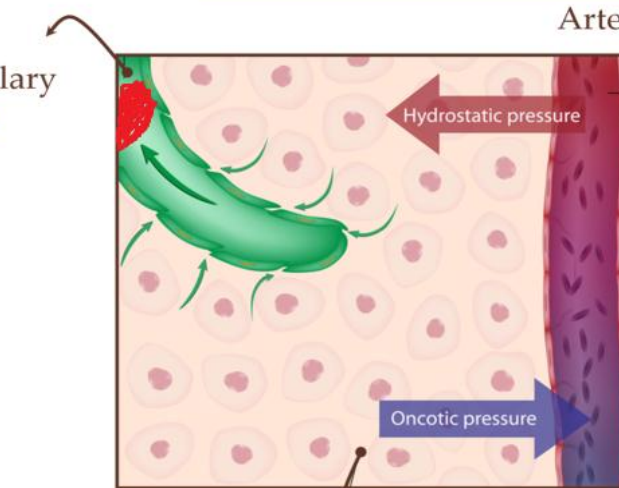


pathogenesis of oedema

Lymphatic Capillary

6.lymphatic obstruction:

- a.congenital
- b.trumatic
- c.inflammation
- d.neoplastic
- e.other



5.increase tissue osmotic pressure

Arteriolar End

1.increase capillary hydrostatic pressure

2.Na and water retention

3.decrease plasma osmotic pressure

4.increase capillary permeability

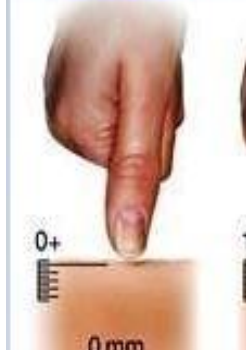
Venular End

INTERSTITIAL SPACE

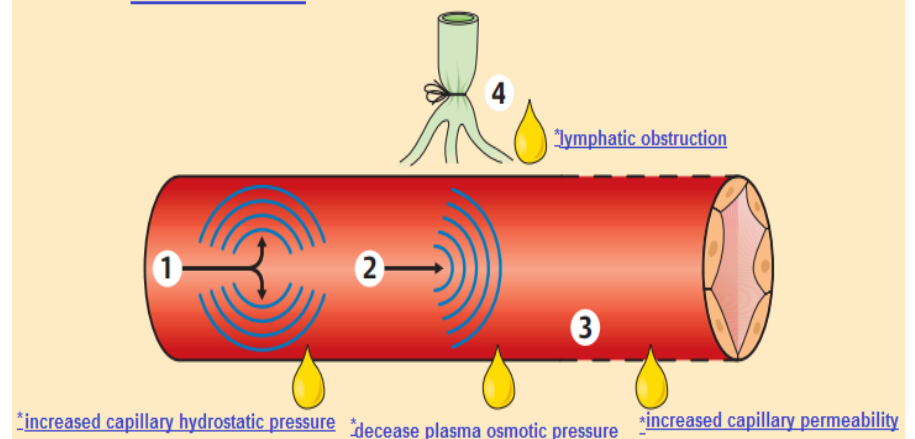
pitting oedema



non pitting oedema



Causes of oedema

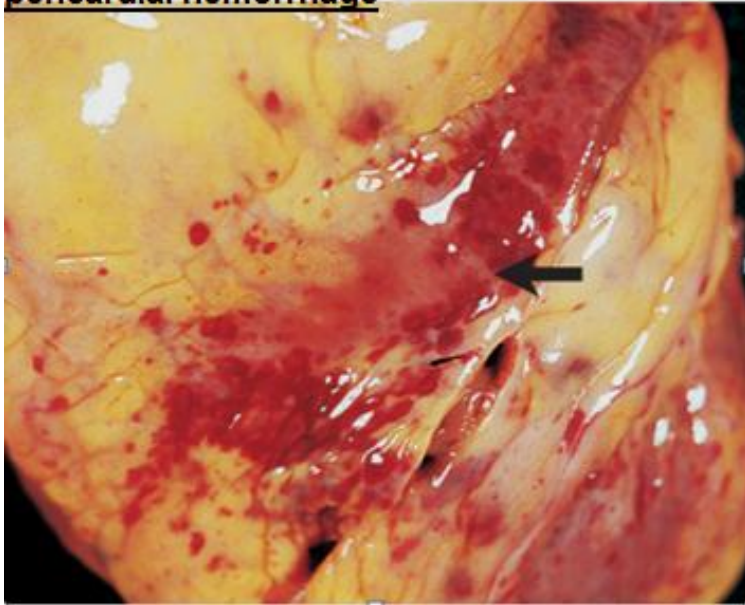


*increased capillary hydrostatic pressure

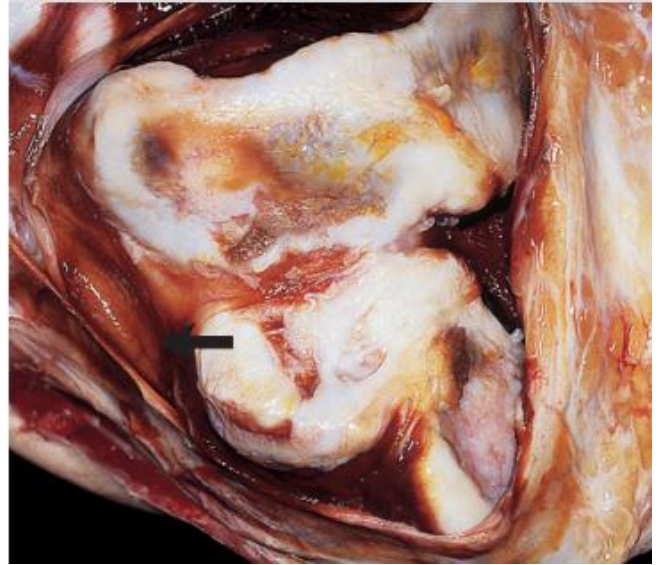
*decrease plasma osmotic pressure

*increased capillary permeability

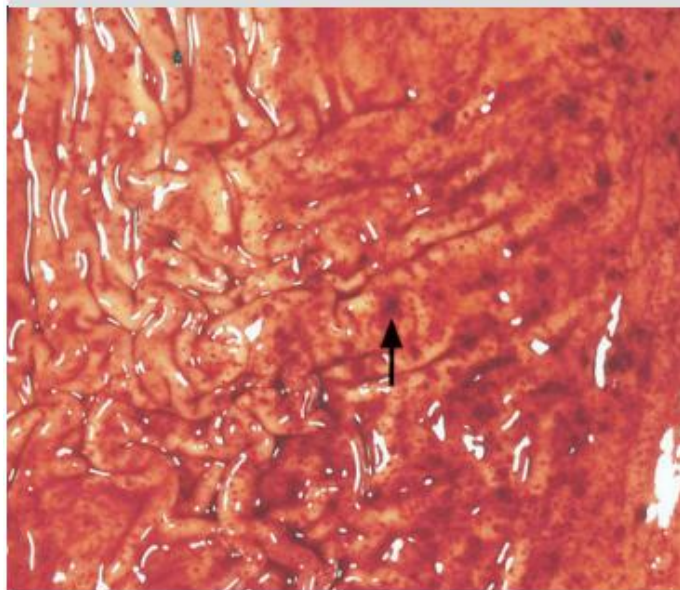
pericardial hemorrhage



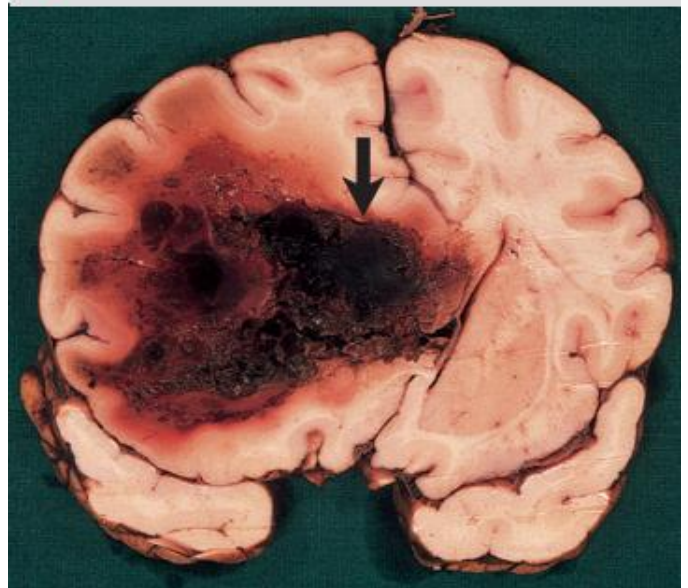
B Hemarthrosis



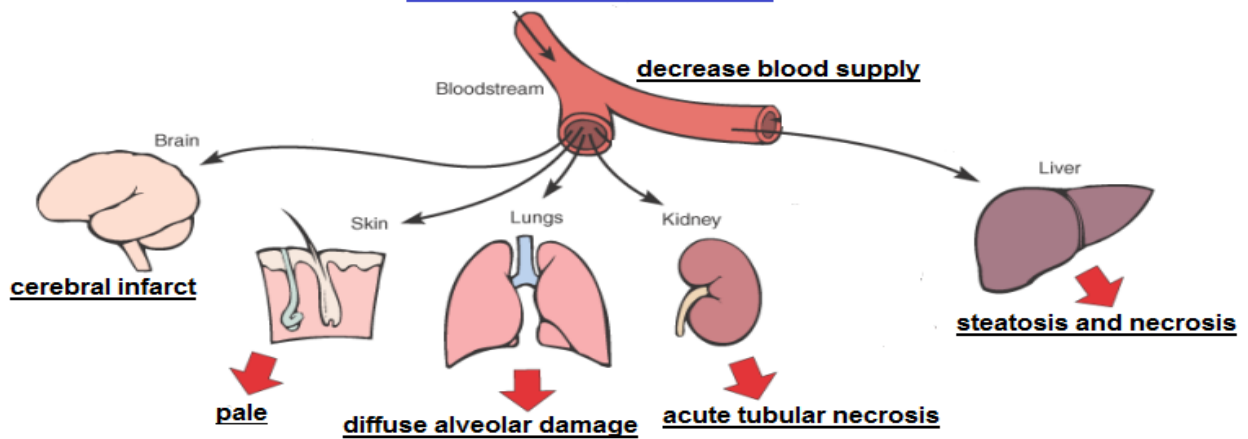
E Petechiae of the gastric mucosa



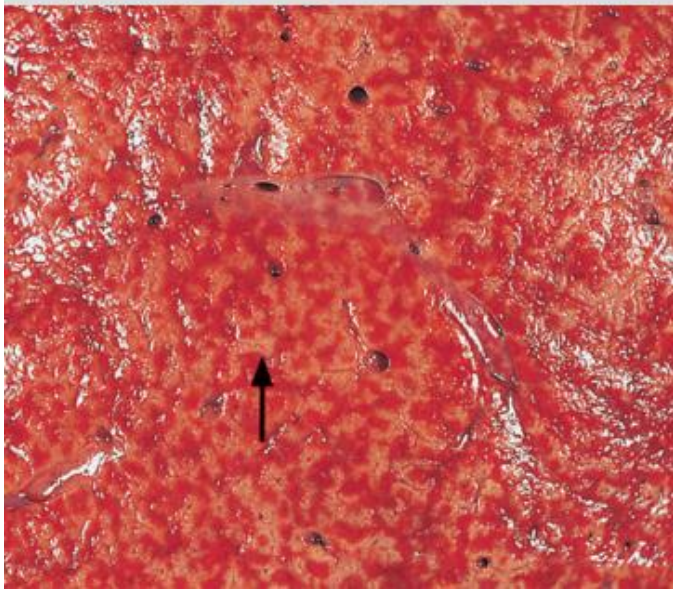
B Massive hypertensive hemorrhage (cerebrum)



post-mortum picture of shock



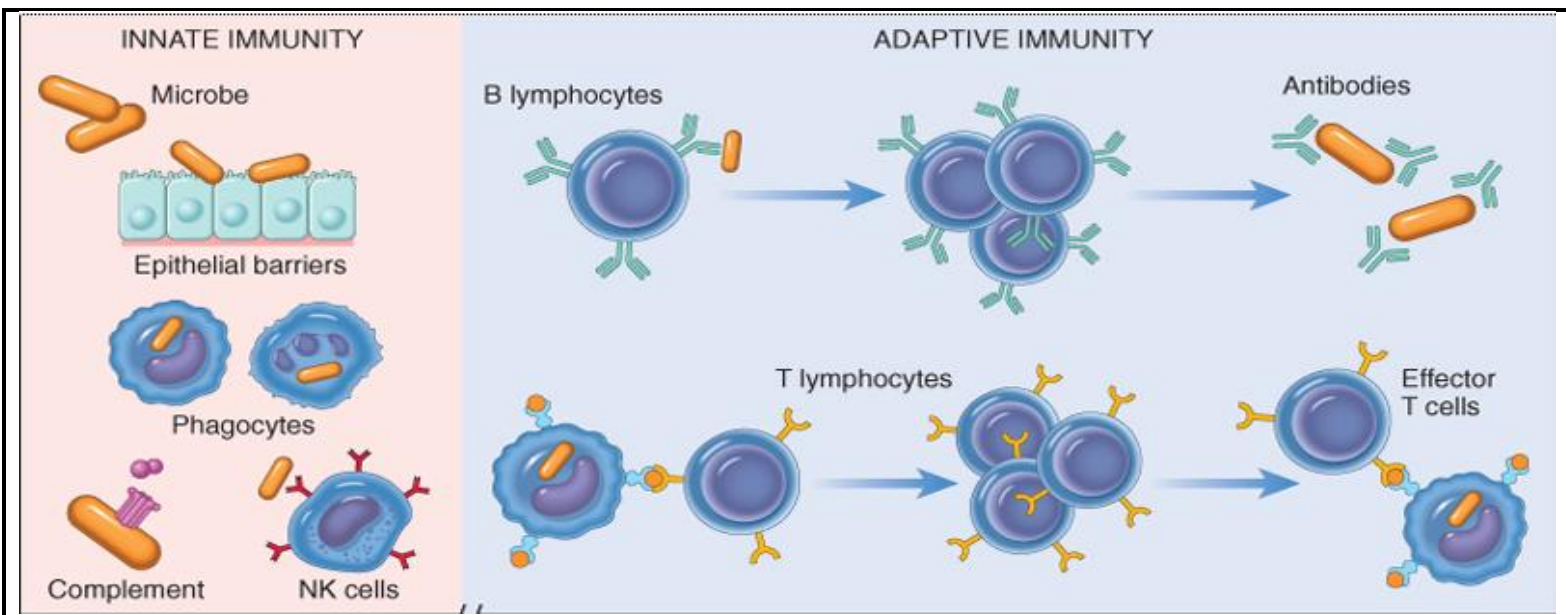
F Shock liver



E Shock kidney (gross)

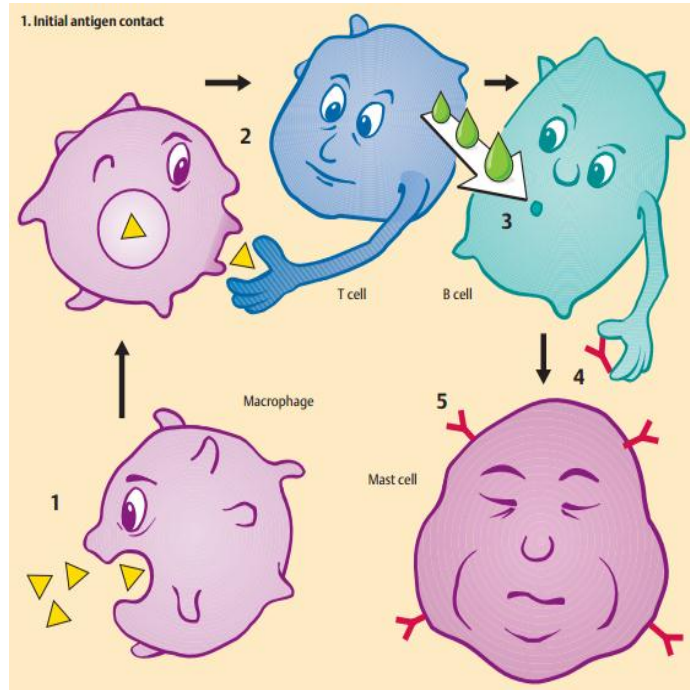


Disorders of immune system

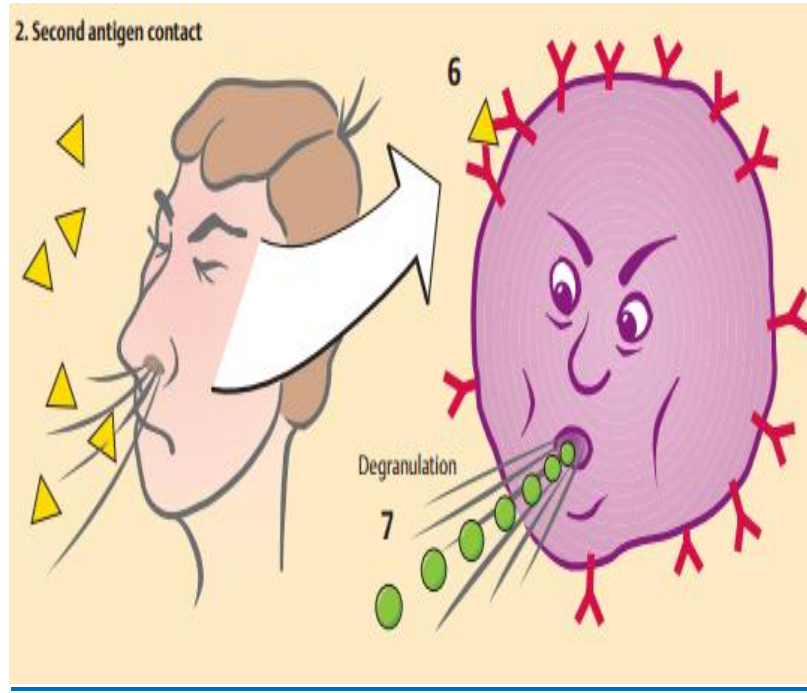


Type 1 hypersensitivity

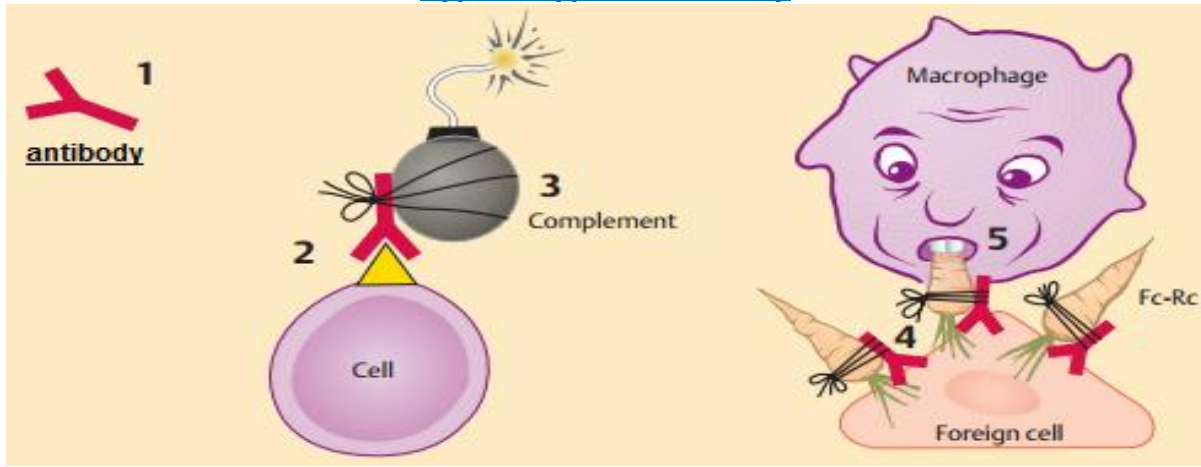
First exposure:



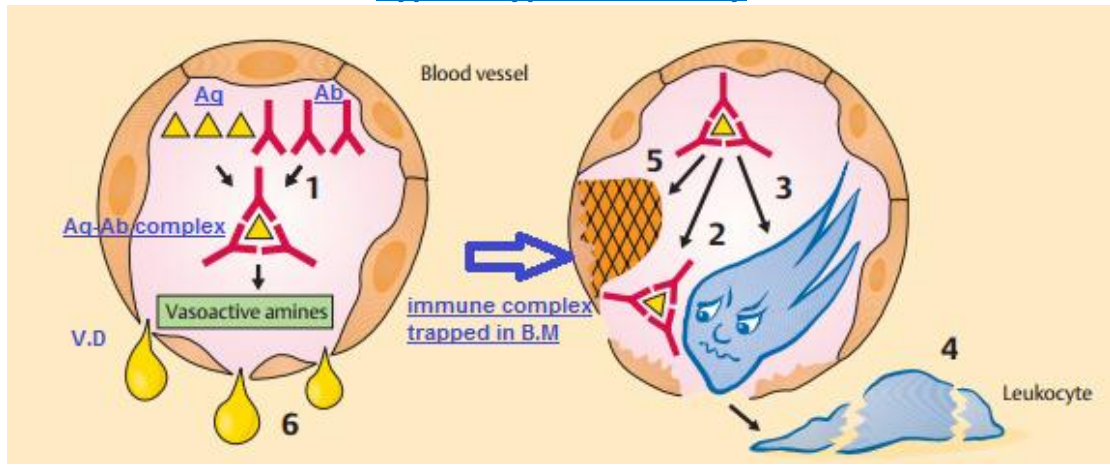
Reexposure:



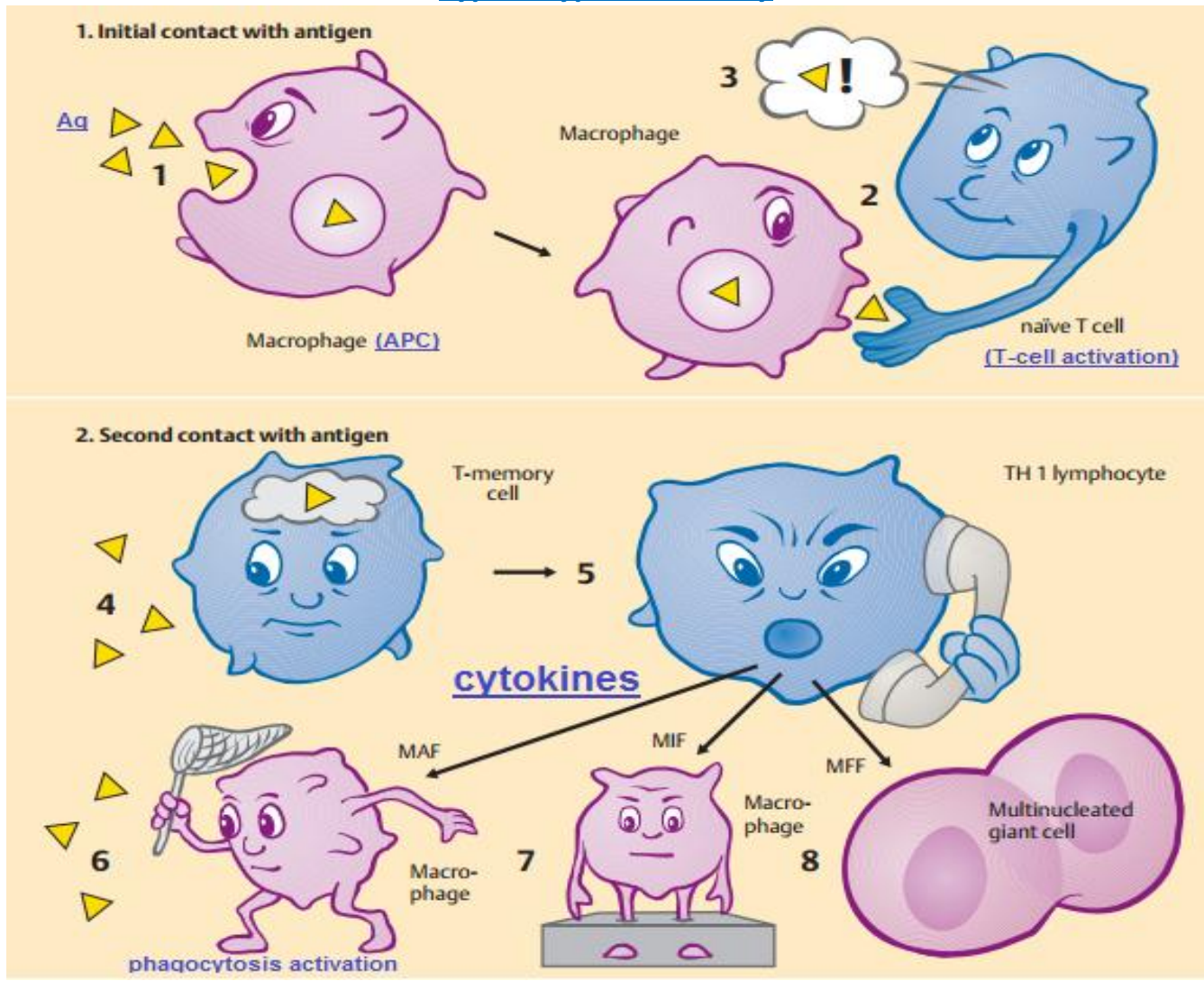
Type 2 hypersensitivity



Type 3 hypersensitivity

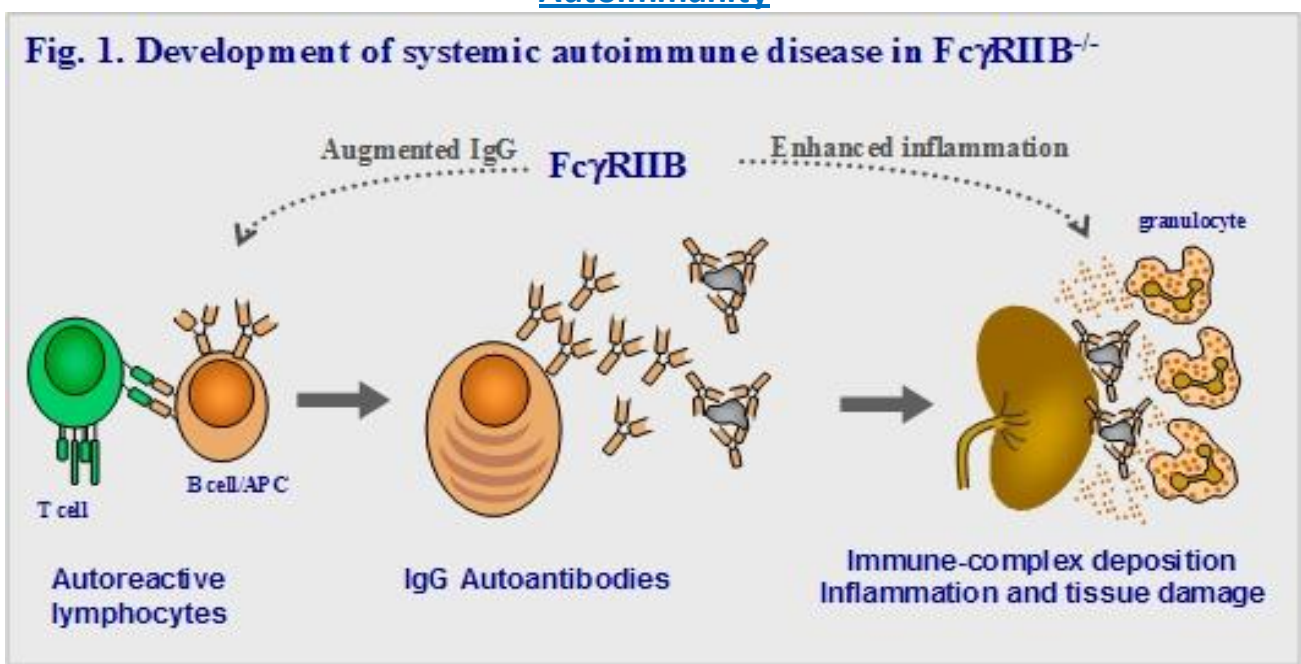


Type 4 hypersensitivity



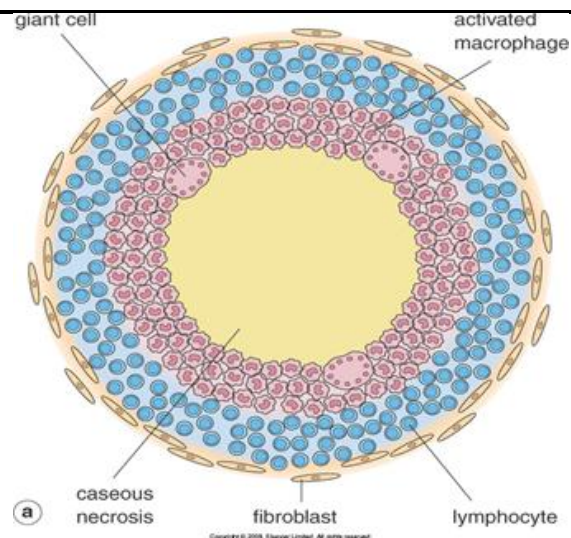
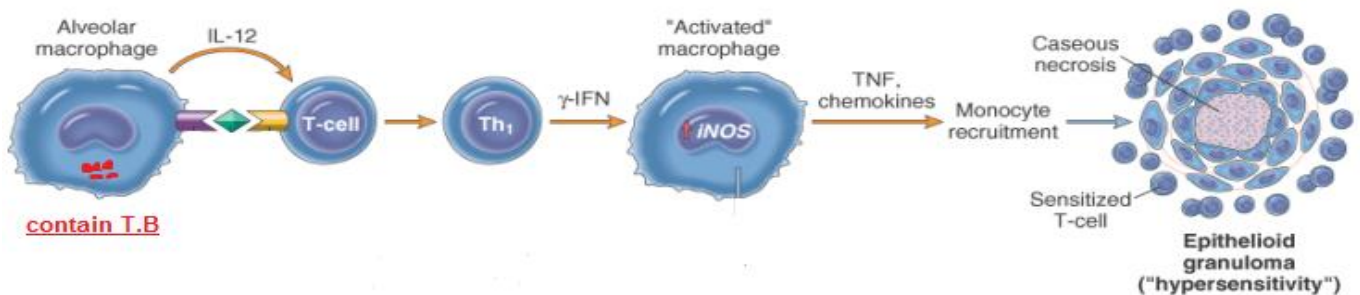
Autoimmunity

Fig. 1. Development of systemic autoimmune disease in $Fc\gamma RIIB^{-/-}$

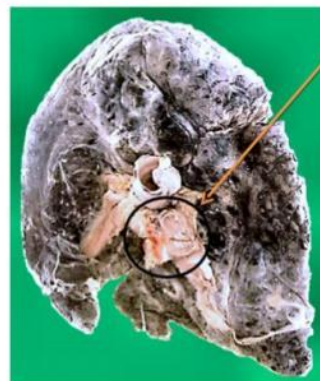
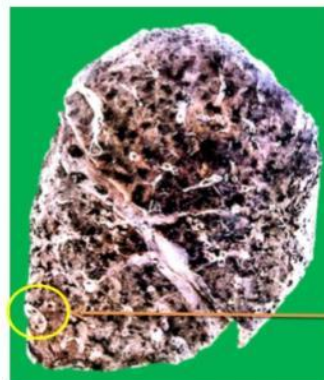


bacterial infection

Pathogenesis of granuloma formation



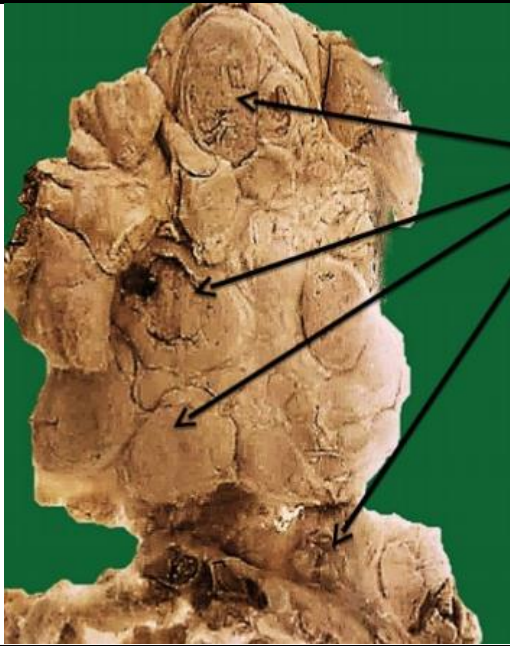
1Y pulmonary T.B



Primary Tuberculous complex of the Lung

- 1) Ghon's focus:
small, round, well-defined, subpleural yellow-white lesion in lower part of the lower lobe
- 2) Enlarged, matted, yellow, caseous hilar lymph nodes

Tabes Mesenterica



- Multiple enlarged mesenteric lymph nodes**
A- matted
B- opaque yellow-grey cut section of caseation necrosis

Tuberculous Pneumonia with Acute Cavity



- 1
 - 2
 - 3
- 1) Acute Cavity:
a- Irregular shape
b- Ragged irregular wall
 - 2) Multiple yellow caseous nodules of tuberculosis
 - 3) Yellow- opaque visceral pleura
- N.B. The lung is consolidated, dark red and firm as indicated by its sharp border

Chronic Fibrocaceous Pulmonary Tuberculosis



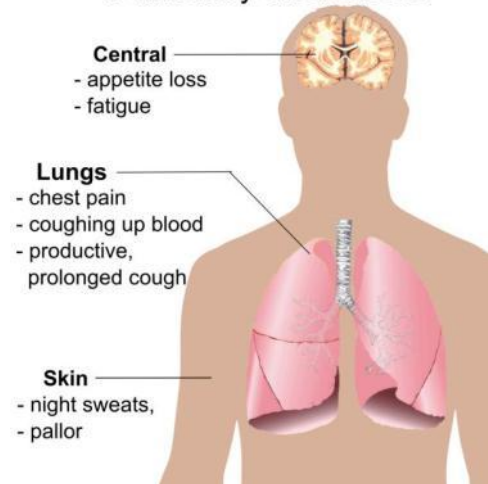
- 1
 - 2
 - 3
 - 4
- 1) Chronic tuberculous cavity:
a- irregular
b- smooth-walled due to fibrosis
c- covered with yellow-white membrane
 - 2) Fibrous cords traversing the cavity and represent fibrosed blood vessels and bronchi
 - 3) Thick fibrous yellow-white pleura
 - 4) The whole right lung is shrunk, yellow, and fibrosed

Miliary Tuberculosis of the Lung

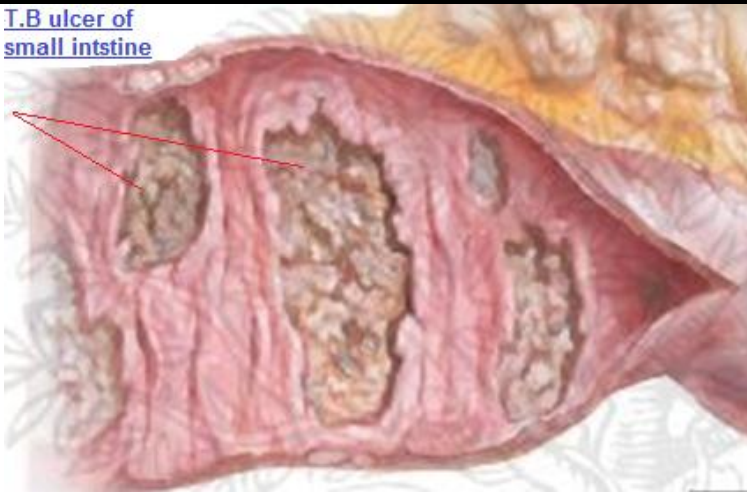


- 1
 - 2
- 1) Multiple, small, yellow caseous foci scattered all over the lung
 - 2) Small anthracotic hilar lymph nodes

Main symptoms of Pulmonary tuberculosis



T.B ulcer of small intestine



Tuberculous Ulcers of Small Intestine

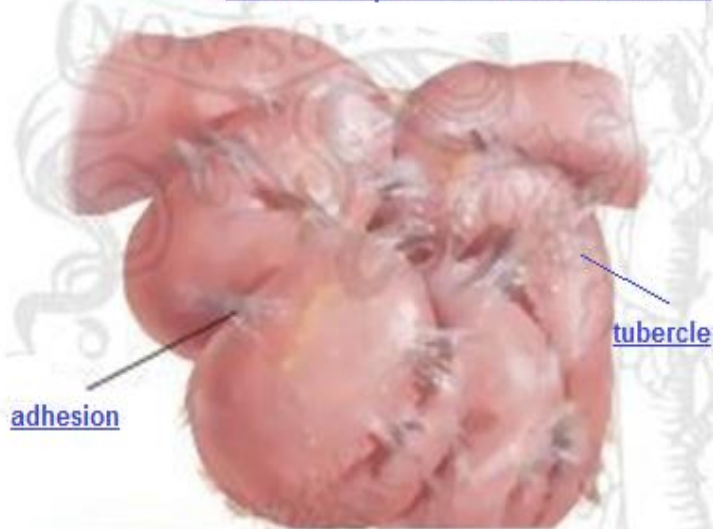


- Multiple
- Round
- Transverse to longitudinal axis of small intestine
- Undermined edge (the pale halo around the center)
- Necrotic floor

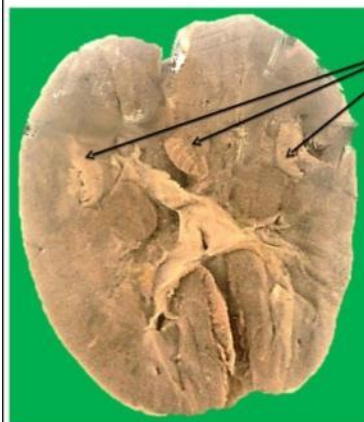
Complication of intestinal T.B



tuberculous peritonitis leads to adhesion

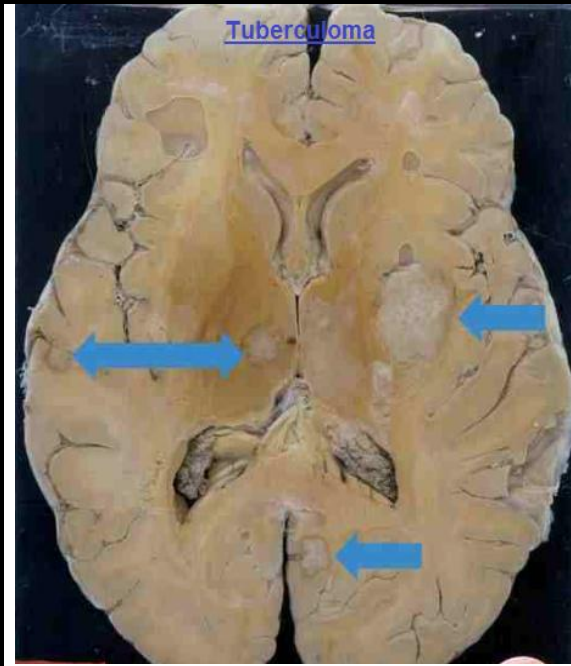
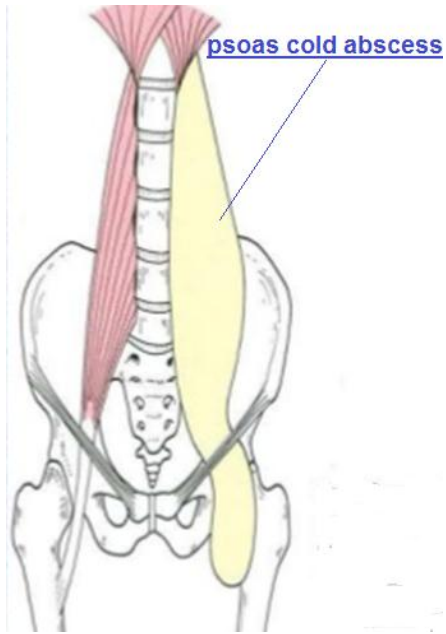
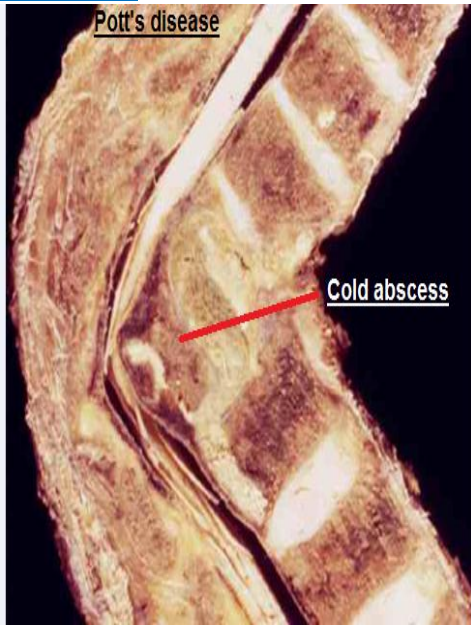


Renal Tuberculosis



Tuberculous infection involving renal papillae with associated papillary caseous necrosis.

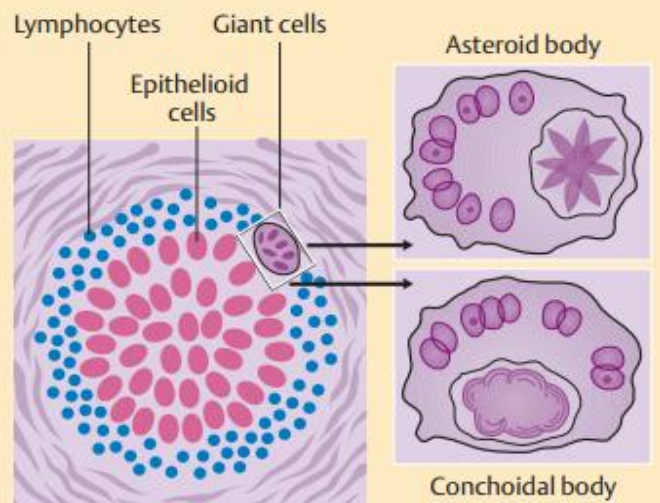
kyphosis



Organs Involved in Sarcoidosis or Sarcoid



A Sarcoid granuloma



S.C nodules in sarcoidosis



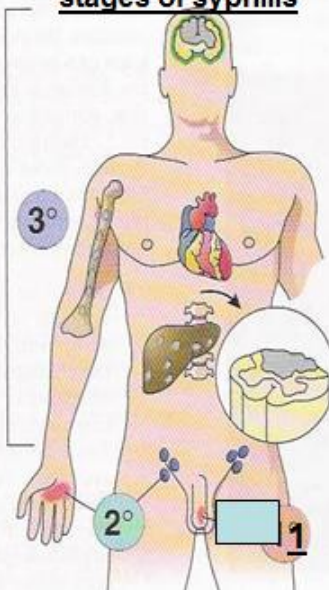
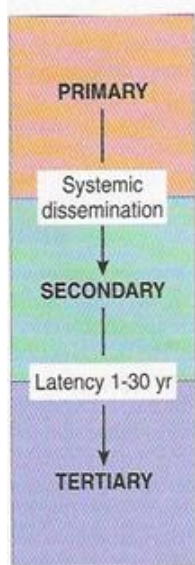
lepromatous leprosy



tuberculoid leprosy



stages of syphilis



Chancere
(male or female
genitalia)

Lymphadenopathy
Rash: palms, soles

Paralytic dementia
Aortic aneurysm
Aortic insufficiency
Tabes dorsalis
Gummas
(widespread)

infective

infective

not infective

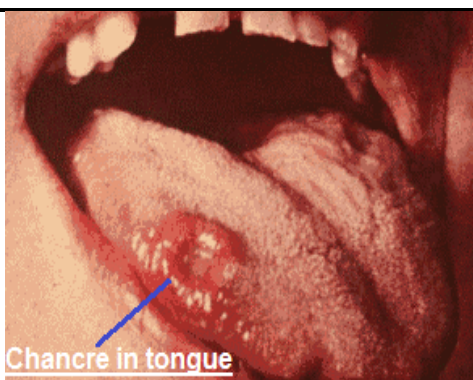
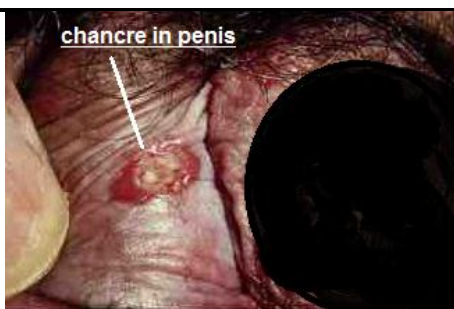
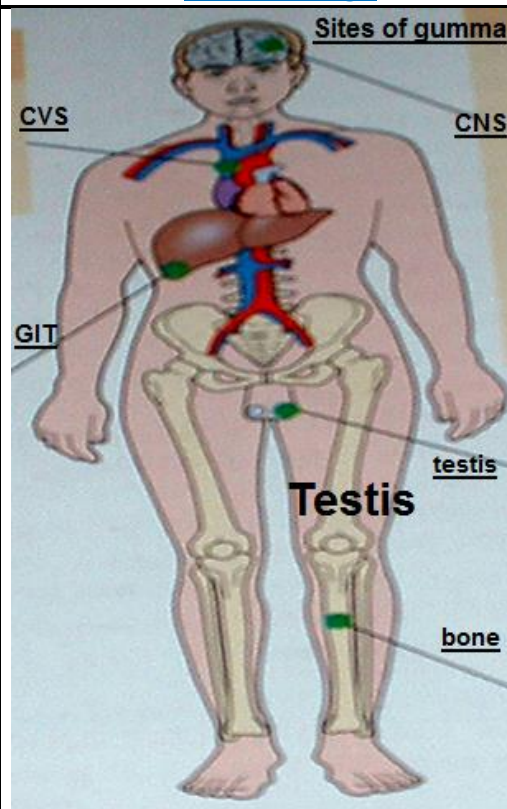
Primary stage



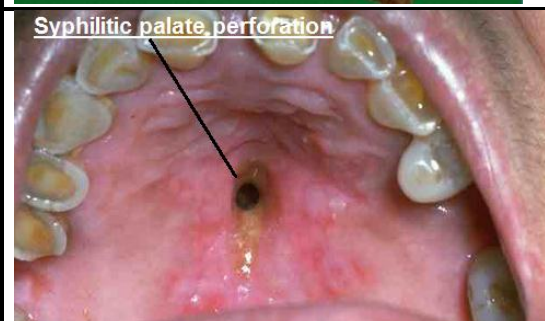
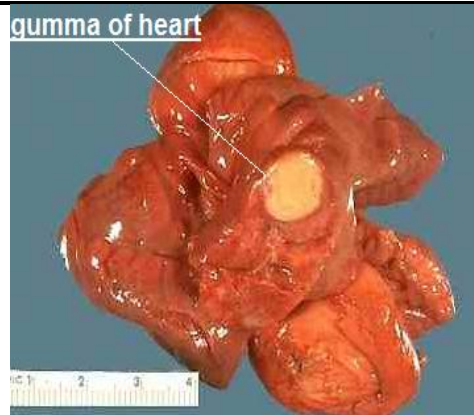
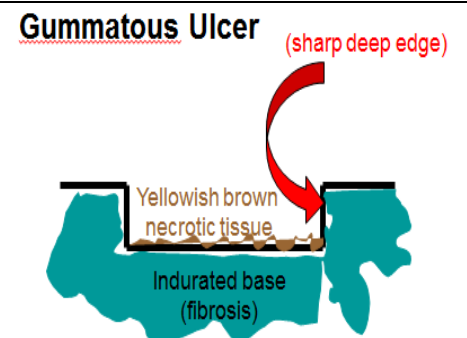
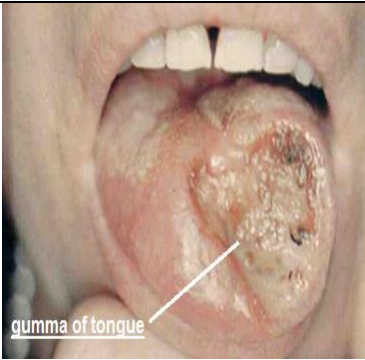
Secondary stage



Tertiart stage

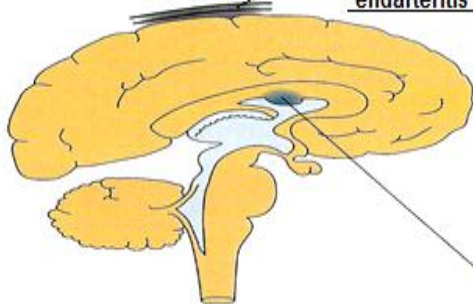


GUMMA (TERTIARY STAGE)

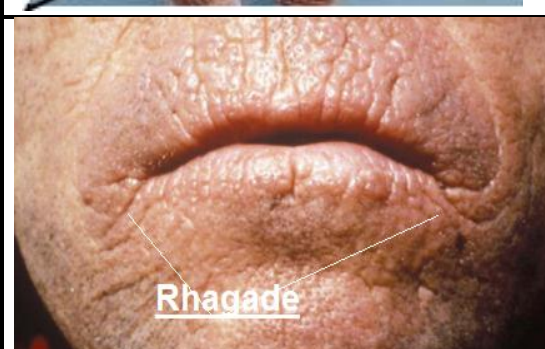
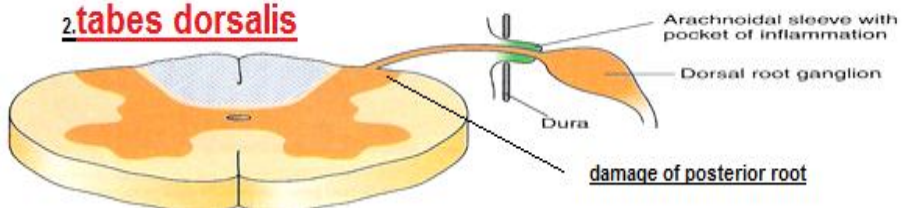


1 menigiovascular syphilis

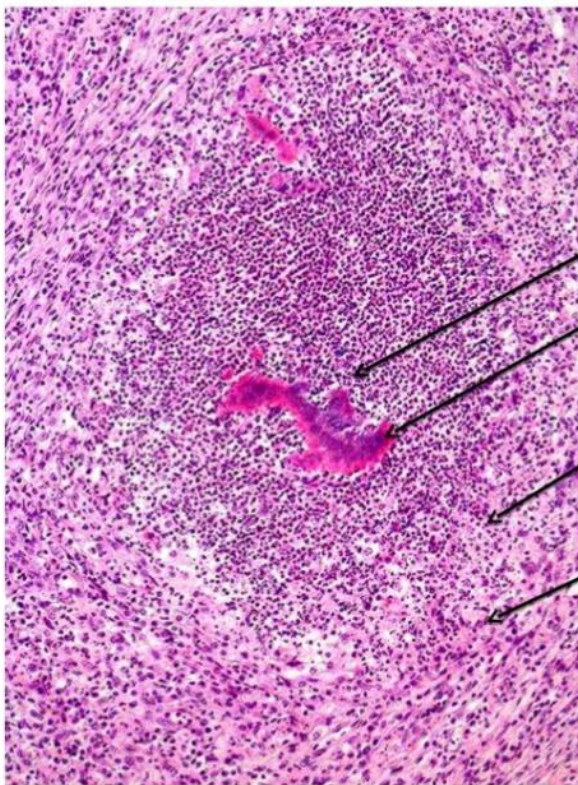
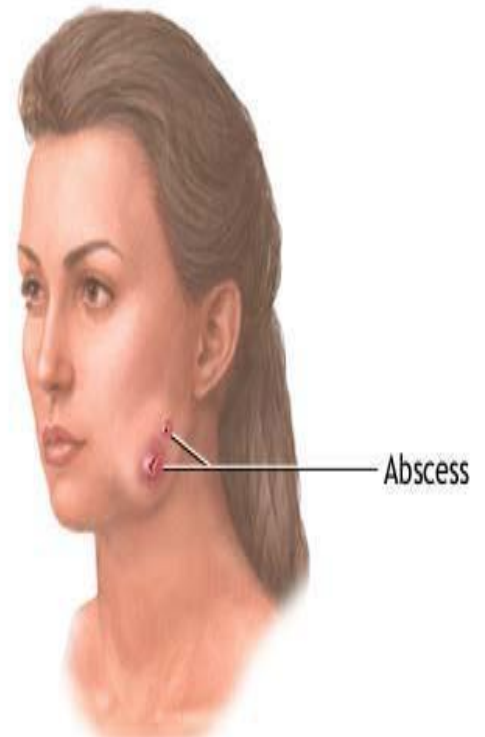
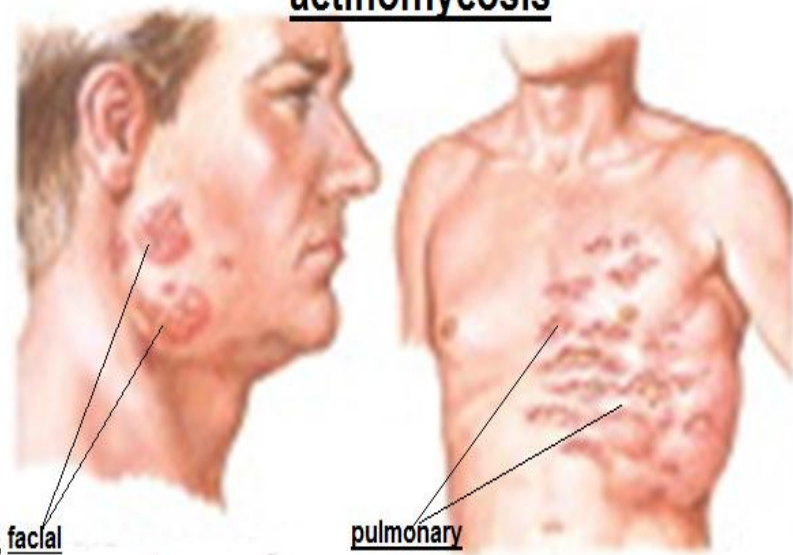
- *thickened meninges
- *endarteritis obliterans



2. tabes dorsalis



actinomycosis



Madura Foot (Actinomycosis)

- 1) Colonies of organism (fungus or bacteria)
- 2) PMNs and pus cells
- 3) Cells of granuloma: epithelioid cells, lymphocytes, plasma cells, eosinophils)
- 4) Areas of fibrosis

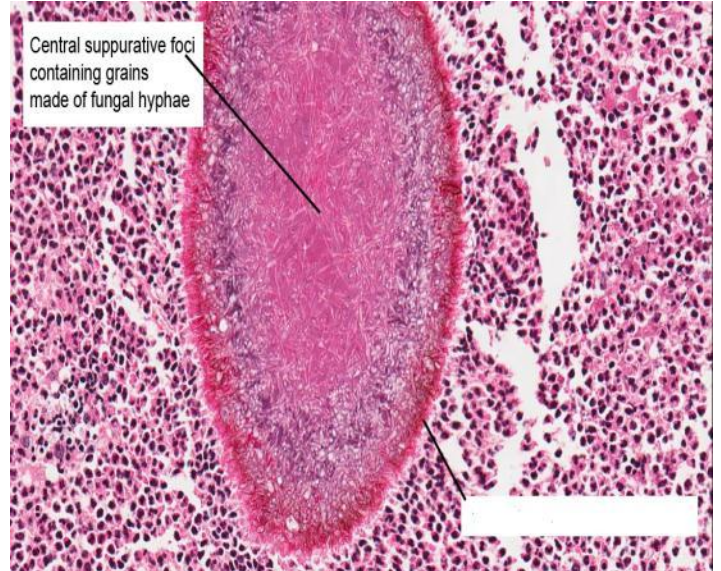
Fungal infection

Madura foot

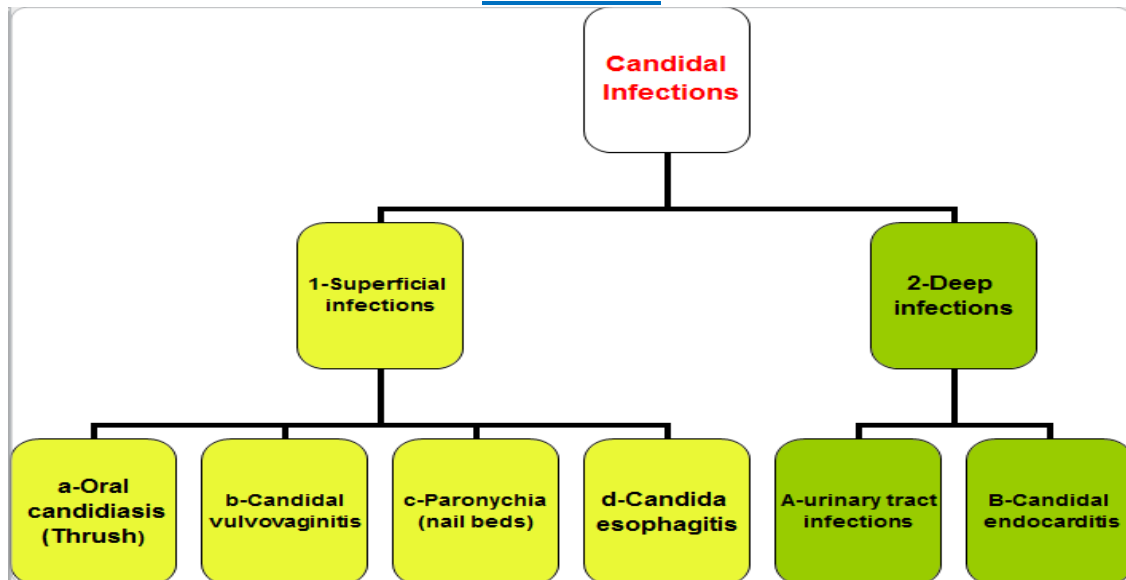
Gross picture



Microscopic picture



Candidiasis



Oral thrush

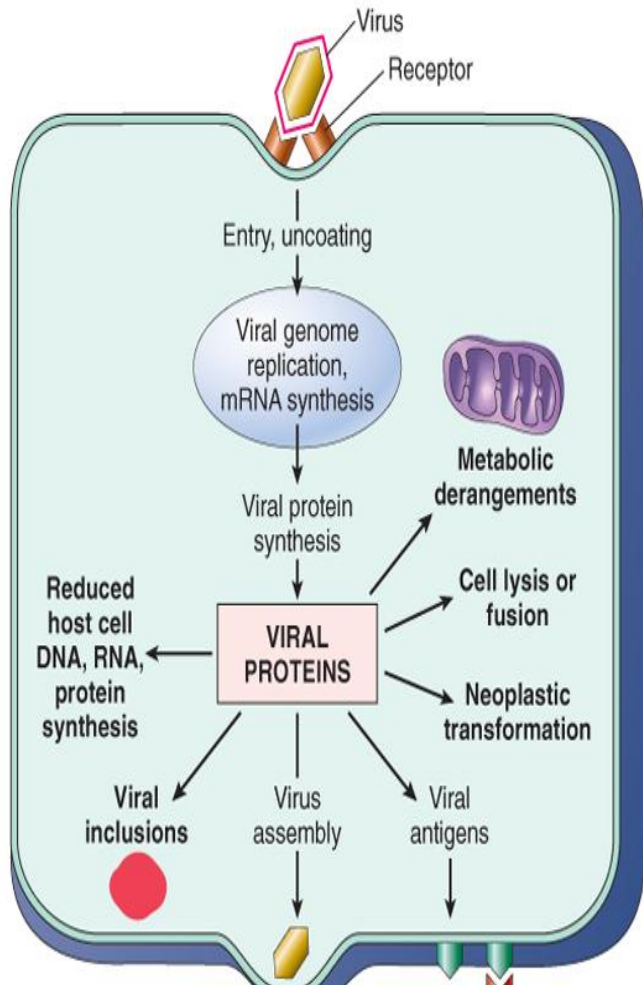


Paronychia



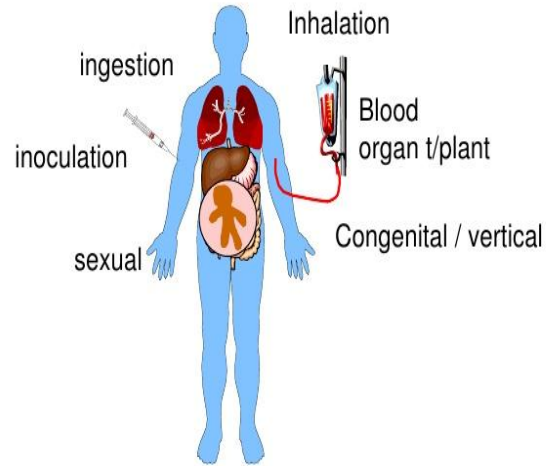
Viral infection

Mechanism of cell injury by virus



Routes of viral infection

Routes of entry:



Tissue reaction to viral infection

a. cellular changes

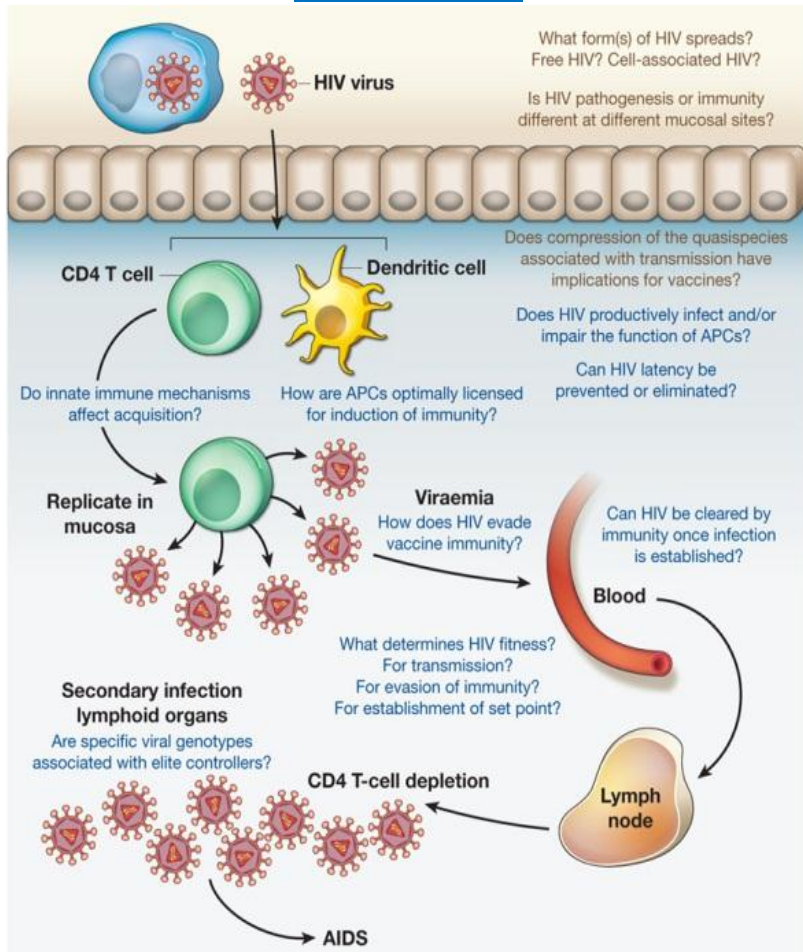
1. inclusion bodies
2. cell degeneration
3. cell necrosis
4. cell proliferation

b. inflammation

1. lymphocytes
2. macrophages

AIDS

Pathogenesis



Pathological features

HIV and AIDS Complications

- Susceptibility to other infections
- Cancers
- Pneumonia
- Death



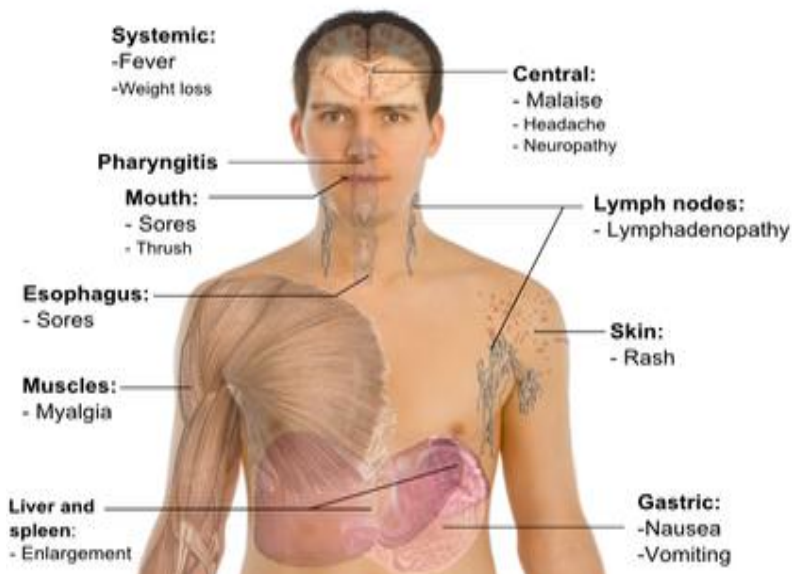
Oral Thrush
Kaposi's Sarcoma

Kaposi's sarcoma



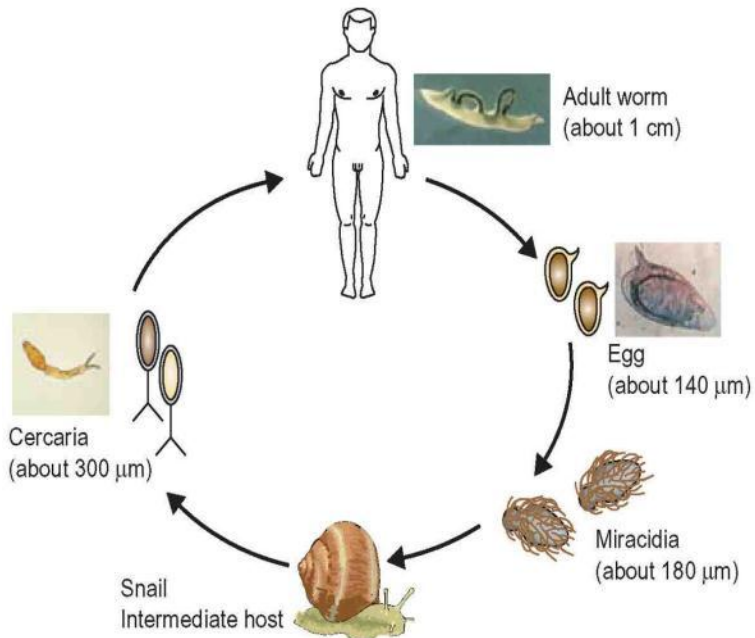
Signs and symptoms

Main symptoms of Acute HIV infection



Parasitic infection

Schistosomal Life Cycle



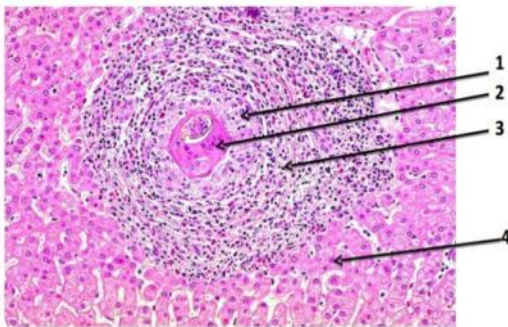
Allergic dermatitis caused by cercaria

Schistosomal dermatitis



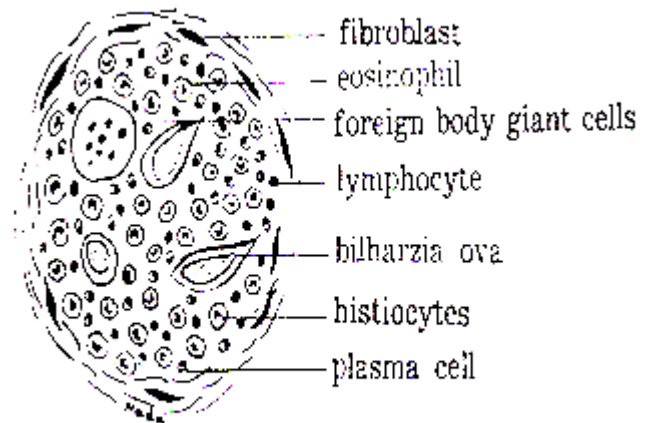
Bilharzial granuloma

Bilharzial Granuloma of the Liver



Bilharzial granuloma formed of:

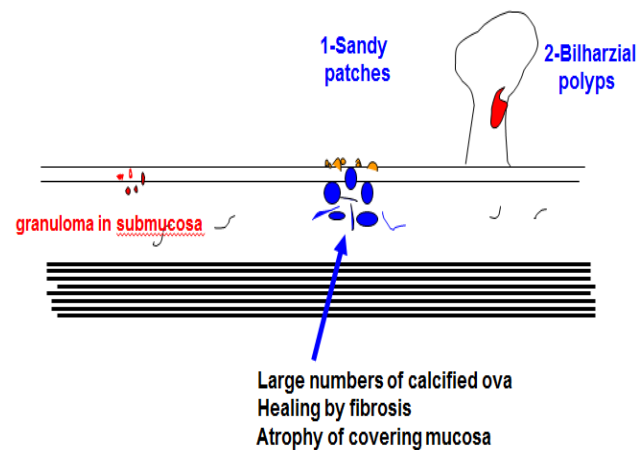
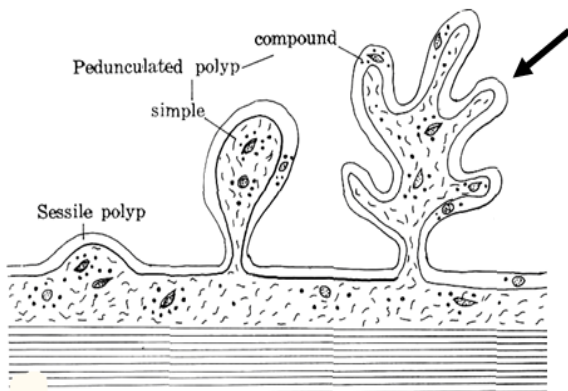
- 1) Epithelioid cells
- 2) Foreign-body giant cells engulfing 2 dead and fragmented bilharzial ova
- 3) Lymphocytes, eosinophils, plasma cells, fibroblasts with early fibrosis
- 4) Normal-looking hepatocytes



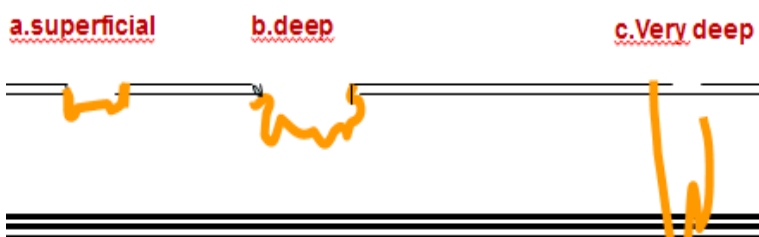
Bilharzioma.

Pathological features

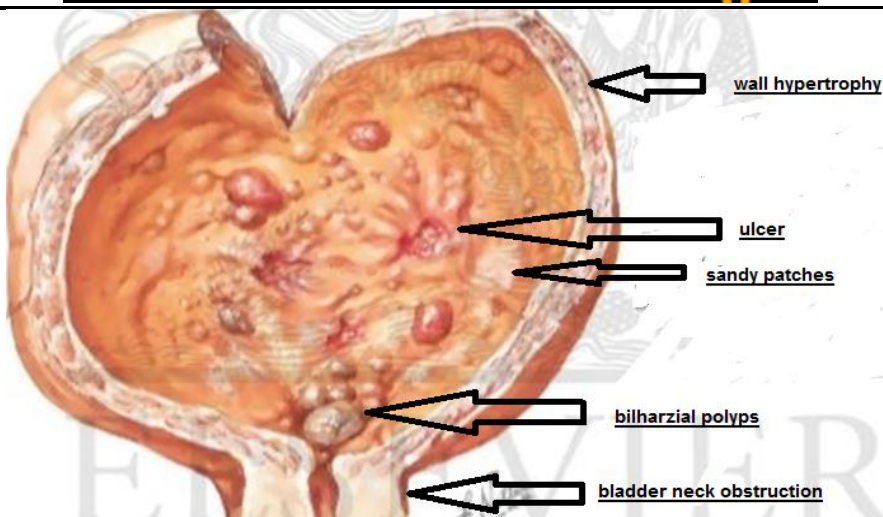
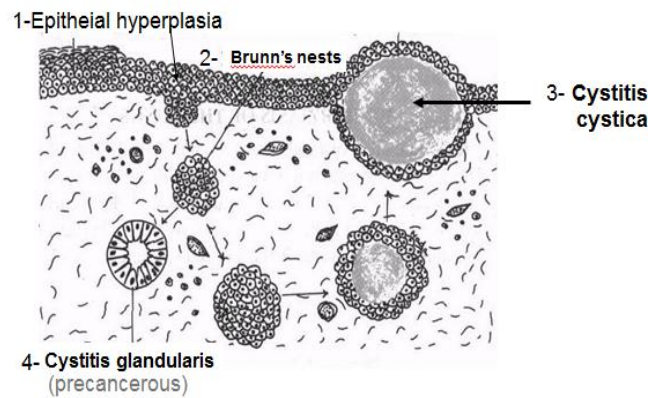
Bilharzial polyp



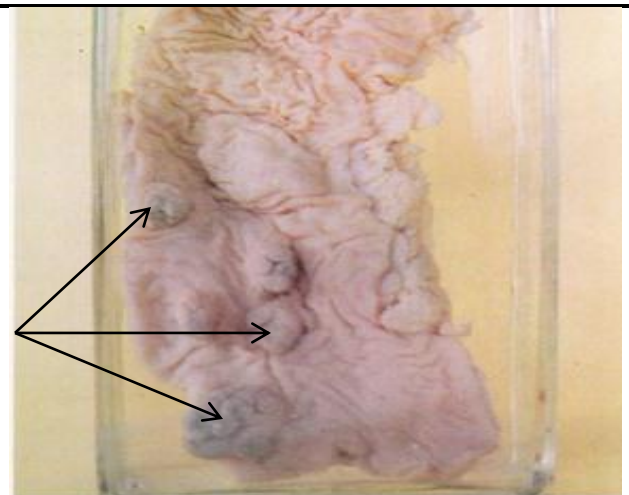
Bilharzial ulcers



Epithelial changes



Bilharziasis of urinary bladder

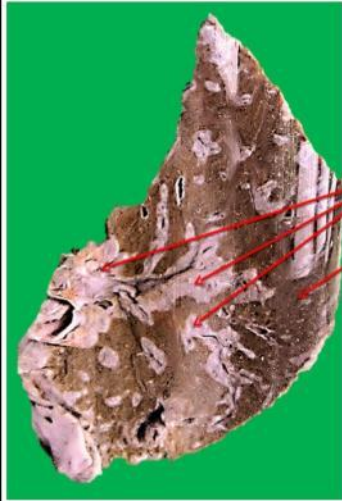


Bilharziasis of large intestine

Bilharziasis of liver

Gross picture:

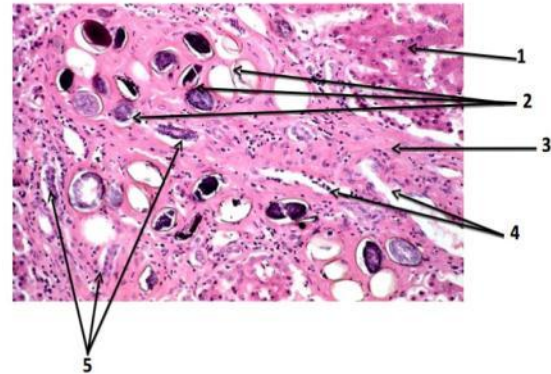
Bilharzial Fibrosis of Liver



- 1) Irregular grey-white areas of fibrosis involving large and small portal tracts
- 2) Irregular granular grey-brown liver surface

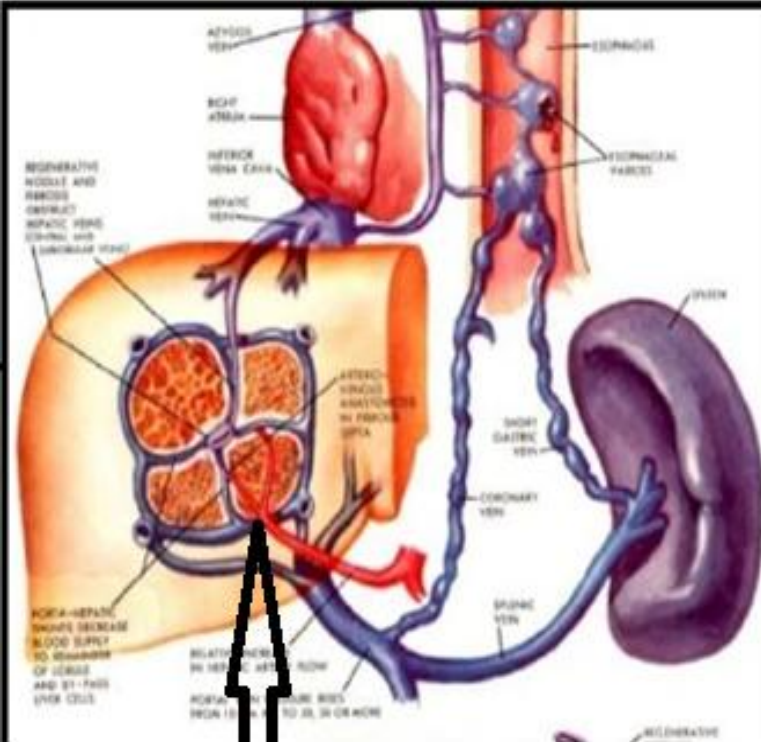
Microscopic picture

Bilharzial Periportal Fibrosis



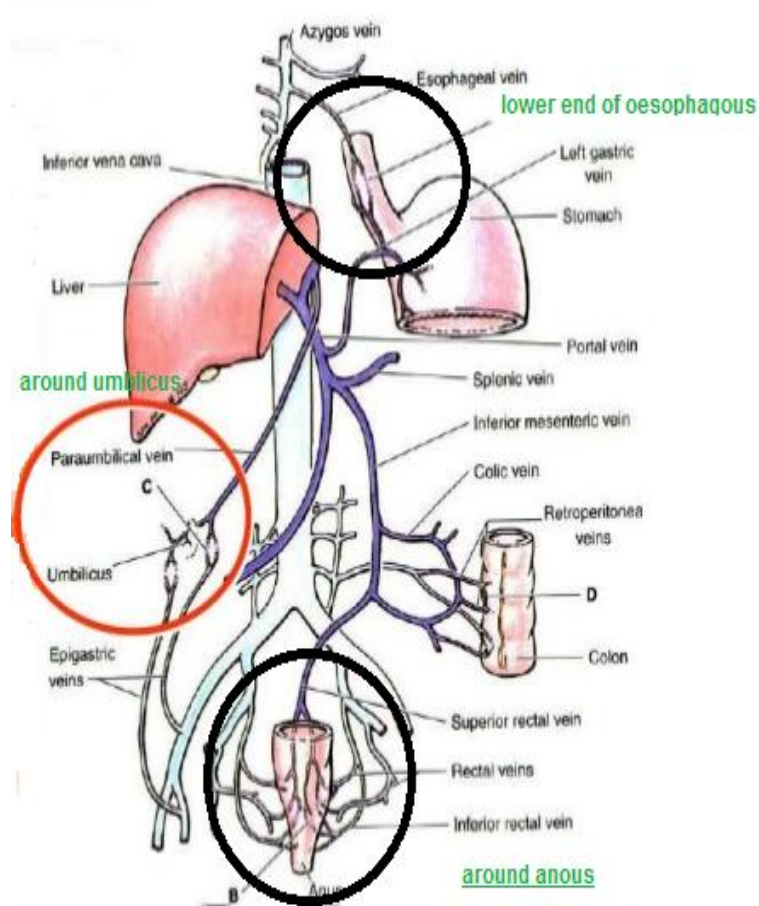
- 1) Normal-looking hepatocytes
- 2) Dead, fragmented, and calcified bilharzial ova
- 3) Fibrosed portal tracts infiltrated with chronic inflammatory cells
- 4) Dilated portal vessels (angiomatoids)
- 5) Proliferating bile ductules

Portal hypertension

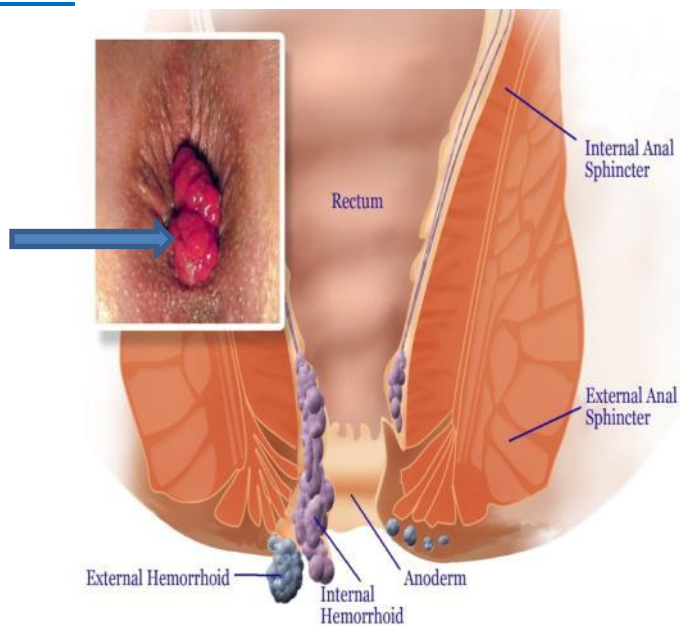


angiomatoid and portal tract fibrosis

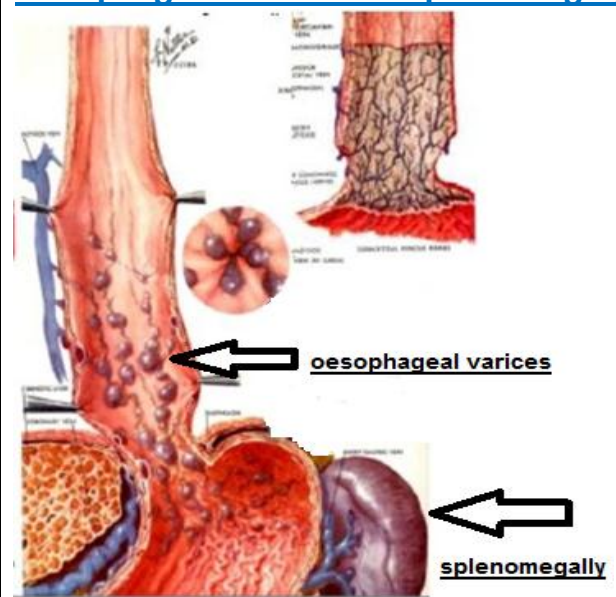
Sites of porto-systemic anastomosis



Piles



Oesophageal varices and splenomegaly



Ascites

ASCITIS LIBRE



Caput medusa



Egyptian splenomegaly Egyptian Splenomegaly (CVC, Spleen)

